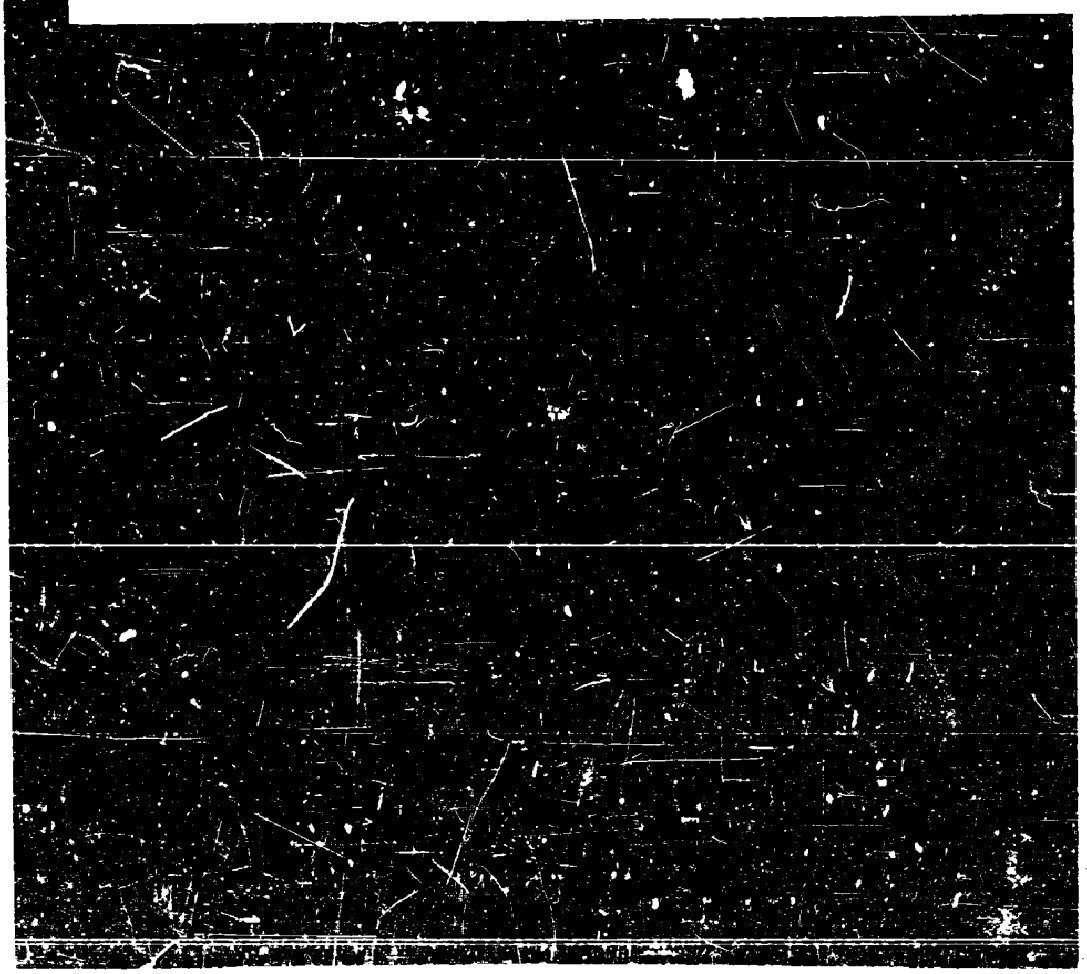
OPERATIONS RESEARCH OFFICE
•
The Johns Hopkins University

Stress in Infantry Combat



Operating Under Contract with the
DEPARTMENT OF THE ARMY

WORKING PAPER

This is a working paper of members of the technical staff of the Tactics Division. Most of the work for this paper was done under former Project Doughboy but was completed under the Infantry Group (code 11 of the new ORO work program).

The objective and scope of the Infantry Group is to analyze the requirements that modern war will impose on infantry, and to recommend systems of organization, tactics, doctrines, and weapons that will optimize infantry effectiveness. This paper (ORO-T-295) deals with one aspect of the over-all objective. The findings and analysis of this paper are subject to revision as may be required by new facts or by modification of basic assumptions. Comments and criticism of the contents are invited. Remarks should be addressed to:

The Director
Operations Research Office
The Johns Hopkins University
7100 Connecticut Avenue
Chevy Chase 15, Maryland

TACTICS DIVISION
INFANTRY GROUP
Technical Memorandum ORO-T-295
Received for Publication: 30 September 1954

Stress in Infantry Combat

by

S. W. Davis
J. G. Taylor

OPERATIONS RESEARCH OFFICE
The Johns Hopkins University Chevy Chase, Maryland

PREFACE

This memorandum is divided into three parts: a summary, a main body, and appendices. The summary and main body are directed toward the military group whose interest is in the tactical significance of stress effects. The appendices contain statistical analyses of data and detailed discussion of the results which, although independent of the main body, will be of most value for those engaged in research on stress.

The results are based on data collected in Korea in 1952 by a team organized and directed by the Operations Research Office, but in addition representing the Office of Naval Research, Naval Medical Research Institute, and Surgeon General's Office of the US Army. Members of the team included: LCDR N. Pace, LT E. L. Schaffer, and LT J. H. Kilbuck, Office of Naval Research Unit No. 1; LCDR D. Minard, Naval Research Institute; Capt E. R. Kolovos and Capt G. H. Longely, Office of the Surgeon General, US Army; and S. W. Davis, F. Elmadjian, L. F. Hanson, and H. S. Liddell, Operations Research Office.

The authors wish to make special acknowledgment of the assistance of Dr. Fred Elmadjian of the Worcester Foundation of Experimental Biology in furnishing some of the analytical material for Appendix A.

CONTENTS

SUMMARY

PROBLEM

To determine the extent to which combat stress affects the tactical usefulness of infantrymen and infantry units.

FACTS

Combat stress can result in ineffective performance of infantrymen. This has been reported by commanders who, in their planning, must make allowances for deterioration in effectiveness of infantrymen as they continue in active combat. Quantifying the effects of combat stress would assist the commander in making proper allowances for stress effects.

As the first step in evaluating stress in combat, a research team went to Korea in the fall of 1952 and examined the physiological and psychological changes that occur as a result of combat stress.

This memorandum is an interpretation of the data obtained in Korea in the light of military effectiveness. Some of these data used here are also described in the preliminary report of the team.* Since that report, there has been further analysis of the samples obtained which has provided additional data. This memorandum is an interpretation of the data which is supported by the symposia held at ORO on stress† and by results reported in the literature.

DISCUSSION

Twelve psychological measures were employed to assess the effects of combat stress on such complex behavior as judgment, memory, abstraction, and the sensitivity of neural mechansims. Twenty-seven physiological measures were used. A majority of these measures were aimed at assessing the effect of combat stress on the function of the adrenal gland. This function has many important effects on the physiological processes and has been shown to be essential for adjustment to stress.

These measures were administered to three groups of infantrymen in Korea. Two of the groups, Able and George Companies, were engaged in active combat of varying intensity and length. Able Company of the 31st Infantry Regiment, 7th Division, was the lead company in an attack on the Triangle Hill area north of Kumhwa on 14 October 1952. The combat was intense and lasted 18 hours

*Operations Research Office, ORO-T-41(FEC), "A Study of Combat Stress, Korea, 1952 (Preliminary Report)."

†Operations Research Office, ORO-T-185, "Fatigue and Stress Symposium," 10 September 1952, and Operations Research Office, ORO-T-256, "The Role of Fatigue and Stress in Military Operations," 2 December 1953.

during which they suffered 61 percent casualties. The group was measured prior to combat and three times after combat—after 12 hours, 5 days, and 22 days. George Company of the 17th Infantry Regiment, 7th Division, occupied from 15 to 20 October a defense position in the same combat area that Able Company had attacked. It took 17 percent casualties during this less intense 5-day combat period. The group was measured twice postcombat--after 12 hours and after 11 days. A third group was comprised of men from three rifle companies of the 15th Infantry Regiment, 3d Division, who had been assigned to a regimental blocking position immediately behind the Main Line of Resistance (MLR) on the central front in Korea. This group, which was not committed to combat, was, however, called upon for frequent patrols and was subjected to heavy artillery fire. It was measured twice (testing times separated by 11 days) and the data were used for purposes of comparison.

The data which proved to be most valuable were those which gave an indication of the activity of the adrenal gland and its effects on body metabolism. The function of the adrenal gland during periods of stress has been the subject of much research. Although there is no single measure of adrenal function, considerable meaning has been attached to the interrelation of many physiological measures in interpreting adrenal function. Some of these measures were observed in Korea and have been used to draw conclusions pertaining to the effects of combat stress.

The investigation of the role of the adrenal gland in stress is progressing rapidly and new information is being added constantly. The effects recorded in this study will not be altered although further research may modify the causal relations discussed.

The conclusions drawn in this study of necessity deal with the alterations of body function and, as such, do not satisfy the final goal, which is to provide the infantry commander with an understanding of the extent to which combat stress affects tactical usefulness of men and units. However, since the groups studied were engaged in typical combat, these conclusions are an important step in answering the ultimate problem since they provide combat information which serves as a firm basis for definition and further experimentation.

CONCLUSIONS

1. There is evidence that being in blocking position behind the MLR does not alter the normal physiological function of combat infantrymen.

2. A short period of intense combat results in a characteristic set of physiological changes for the average infantryman (a high degree of adrenal responsiveness, increased protein destruction, and a shift in the balance of body salts).

3. A period of prolonged, less intense combat results in a different set of physiological changes in the average infantryman (a low degree of adrenal responsiveness, normal protein destruction, and a shift in the balance of body salts opposite to that observed in short, intense combat).

4. Although the response of both groups can be considered normal stress responses, that of the short, intense group is characteristic of the initial or adaptation phase of the stress reaction, and that of the prolonged, less intense group is characteristic of the second or resistance phase of the stress reaction.

5. Men exposed to the same combat reach the period of maximal physiological adaptation to stress at different times.

6. Time taken to recover to normal physiological levels after intense combat of 18 hours is approximately 6 days; after less intense combat of 5 days it takes approximately 13 days.

7. Based on physiological function alone, combat of a high level of intensity is more costly than combat of a low level of intensity in that recovery time is longer in proportion to the duration of the stress.

8. Based on physiological function alone, the initial period of combat is more costly than continuing in combat past the initial period in that recovery time is longer in proportion to the duration of the stress.

STRESS IN INFANTRY COMBAT

INTRODUCTION

Objectives

A commander must consider the degradation of effectiveness of his men as they continue in active combat, just as he considers and compensates for the degradation of performance of his weapons. The course of weapons deterioration as a function of rate and amount of fire is well defined, and correction for this deterioration has become standard procedure. The course of the deterioration of performance of infantrymen due to the stress of active combat has not been determined. Therefore, in making allowances for performance degradation, the infantry commander has had to rely on intuition and rules of thumb based on inadequate information.

As part of a consideration of infantry combat effectiveness, it is important to be able to determine the extent to which combat affects the infantryman's performance. The course of performance changes during combat and the period of recovery should be traced using indices readily available to the commander. Given such knowledge of deterioration to supplement his consideration of previous performance, status of training, etc., the infantry commander would be better able to determine the length of time a company can remain effective in combat, which of several units is in best condition to accomplish a given mission, and at what time a relieved unit is ready to be used again.

Background

Some of the foremost scientists working on problems of stress attended a symposium at ORO to help this organization form a logical plan for determining the relation between combat performance and combat stress. They concluded that "present knowledge of the fundamental underlying mechanisms of fatigue and stress is inadequate to define the degree of impairment, establish permissible limits of deterioration, predict imminence of complete collapse, and describe the interrelation of stresses and tolerance limits under varying conditions." And recommended that "description and definition of the nature of the problem and identification of important stress factors and fatigue reactions in combat situations should probably be made under combat conditions."[1(4)]* Based on this recommendation, a research team of 13 went

*Superior numbers refer to the reference section at the end of this memorandum; numbers in parenthesis refer to page numbers when cited with the text reference.

[illegible] to study stress under combat conditions. A detailed description of the methods of measurement, the raw data obtained, and a preliminary analysis of portions of these data are reported in ORO-T-41 (FEC).[2]

Following the return of the team from Korea, the results of the study were presented at a second symposium composed of representatives of military and civilian research organizations, in order that their research experience could be brought to bear in interpreting the findings.[3]

The analysis of the data obtained in Korea, in the light of information gained from the symposia and a detailed survey of the literature, is the major subject of this memorandum and provides some important facts regarding (a) the different physiological condition of groups engaged in combat of varying intensity and length, (b) differences among individuals in each of the groups, and (c) time taken to recover from combat of different intensity and length. Obtaining information pertaining to these problem areas does not satisfy the final goal, i.e., to provide the infantry commander with an understanding of the extent to which combat stress affects performance or with readily available indices of performance changes with combat stress. However, lacking basic combat data at the outset, these facts are an essential step toward satisfying this ultimate goal.

For purposes of study the total stress reaction has been divided into two general categories: physiological and psychological. The physiologists are concerned with alterations of bodily functions with stress. Measures in this area are readily quantifiable and are prerequisite to a complete understanding of the body's total reaction to stress. The psychologists have emphasized the study of alterations of complex behavior with stress. Measures in this area are not so readily available or quantifiable. Since maintenance of suitable behavior patterns is important to military effectiveness, however, a knowledge of behavioral changes with stress is essential.

The team which went to Korea to study reactions to combat stress represented fields of physiology, psychology, medicine, and psychiatry. The team operated in Korea from a laboratory which was established at the 8063 Mobile Army Surgical Hospital (MASH) supporting the 7th Division on the central portion of the Main Line of Resistance (MLR).

Psychological Measures

The psychological measures used by the team were selected to measure the effects of combat stress on such complex behavior as judgment, memory, abstraction, and the visual and auditory critical fusion frequencies. It is these measures which the symposium[1] and a survey of the literature[4] suggested would be the most affected when the tolerance limits of the higher functions of the central nervous system are being exceeded in combat. Judgment, understanding, and similar higher mental functions are assumed to be fundamental to the maintenance of effective combat performance. The actual tests were for the most part established tests designed for measuring the behavior factors outlined above, although in some instances the standards set by the authors were modified to meet field conditions.

Physiological Measures

The majority of the physiological measures selected by the team were aimed at assessing the effect of combat stress on the functioning of the adrenal glands (see Fig. 1). The adrenal glands, located just anterior to the kidneys, are important in protecting the individual during stress, for it has been found that without this gland the organism cannot withstand or make adjustment to

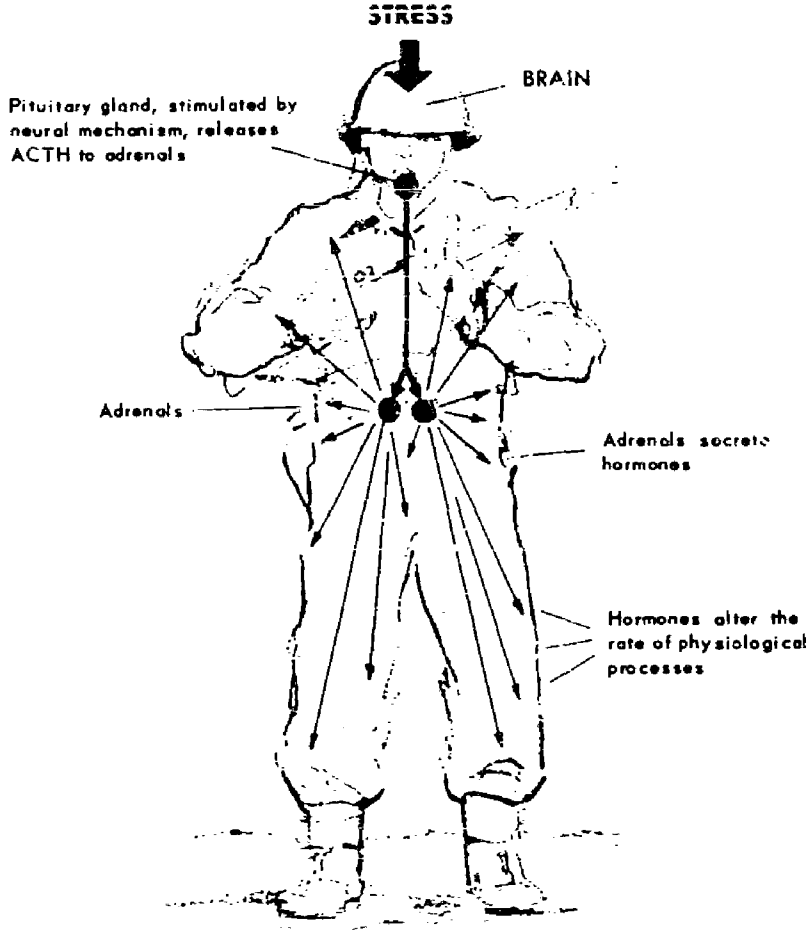


Fig. 1—Schematic Representation of Pituitary Adrenal Function in Response to Stress

stress. The pituitary gland, which is stimulated by stress acting on the nervous system, secretes adrenocorticotrophic hormones (ACTH), which in turn stimulate the adrenal glands. When the adrenal glands are stimulated by ACTH they secrete adrenal hormones or corticoids, the principal one being Compound F. Although these hormones do not create any new functions for the body, they do alter the rates of physiological processes important in the body's reaction to stress.

The measurements of adrenal cortical function used in this study are of two kinds:

(a) The amount of actual hormone secreted by the adrenal cortex (corticoids) which appear unchanged in the urine and the metabolic end products of the adrenal hormones which also appear in the urine. Portions of the corticoids, including Compound F, secreted by the adrenal cortex are excreted unchanged in the urine. In addition some of the corticoids are changed as they go through the body and these metabolic end products of the corticoids are excreted in the urine. One group of these corticoids and their end products were measured by determining the Porter-Silber (P.S.) chromogens in the urine. A different group of these corticoids and their end products were measured by determination of 17 ketosteroids (KS)*. The presence or absence of the P.S. chromogens and 17 KS in the urine have been related to adrenal function. The increase or decrease in the function of the adrenal gland has been related to the quantity of these compounds present in the urine. Typically the P.S. chromogens and 17 KS excreted in the urine increase above normal levels following exposure to stress. After 2 to 3 days of severe stress, such as surgery or fractures,

*The active adrenal cortical hormones are steroids of Pregnane series (Figs. a and b). The metabolites of these hormones which appear in the urine may be classified into C 21 steroids, e.g., that shown in Fig. c, and C 19 steroids, e.g., that shown in Fig. d.

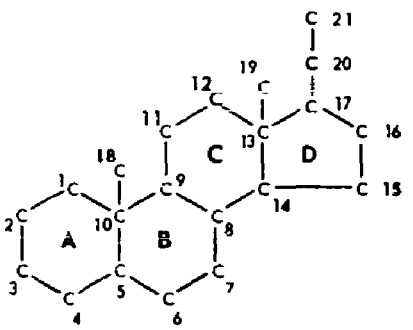


Fig. a; Schematic Diagram of a Typical Pregnane Steroid

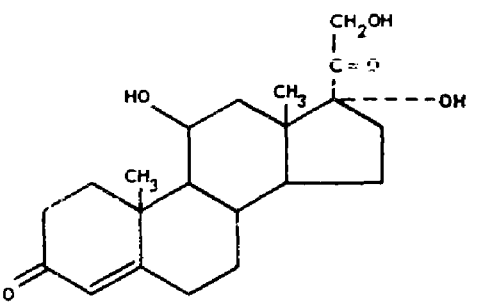


Fig. b; Compound F: a Pregnane Steroid

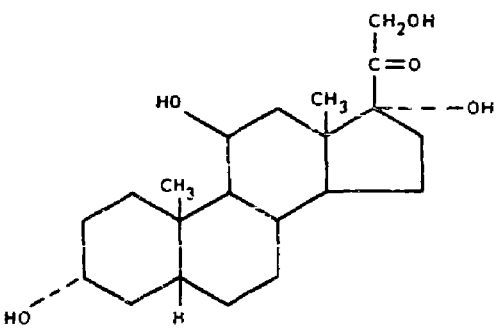


Fig. c; C 21 Steroid: a Reduced Form of Compound F

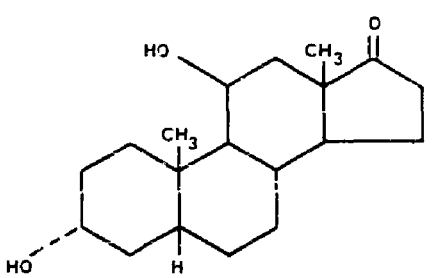


Fig. d; C 19 Steroid: an Oxidized Form of the Compound Shown in Fig. c

The C 21 steroids were estimated by the Porter-Silber reaction. This titer measures Compound F and its metabolites where reduction has taken place in Ring A. The reactive portion of the molecule consists of the 17 hydroxy configuration in addition to the alpha ketol side chain (Fig. c).

The C 19 steroids estimated were 17 KS (Fig. d) with the Zimmerman reaction, which gives a titer of a group of compounds which are in largest portion end products of adrenal cortical hormone metabolism. The reactive portion of the molecule is the ketone on carbon 17. The alpha ketol side chain has been oxidized to the 17 KS, or the 17 KS come directly from the adrenal gland.

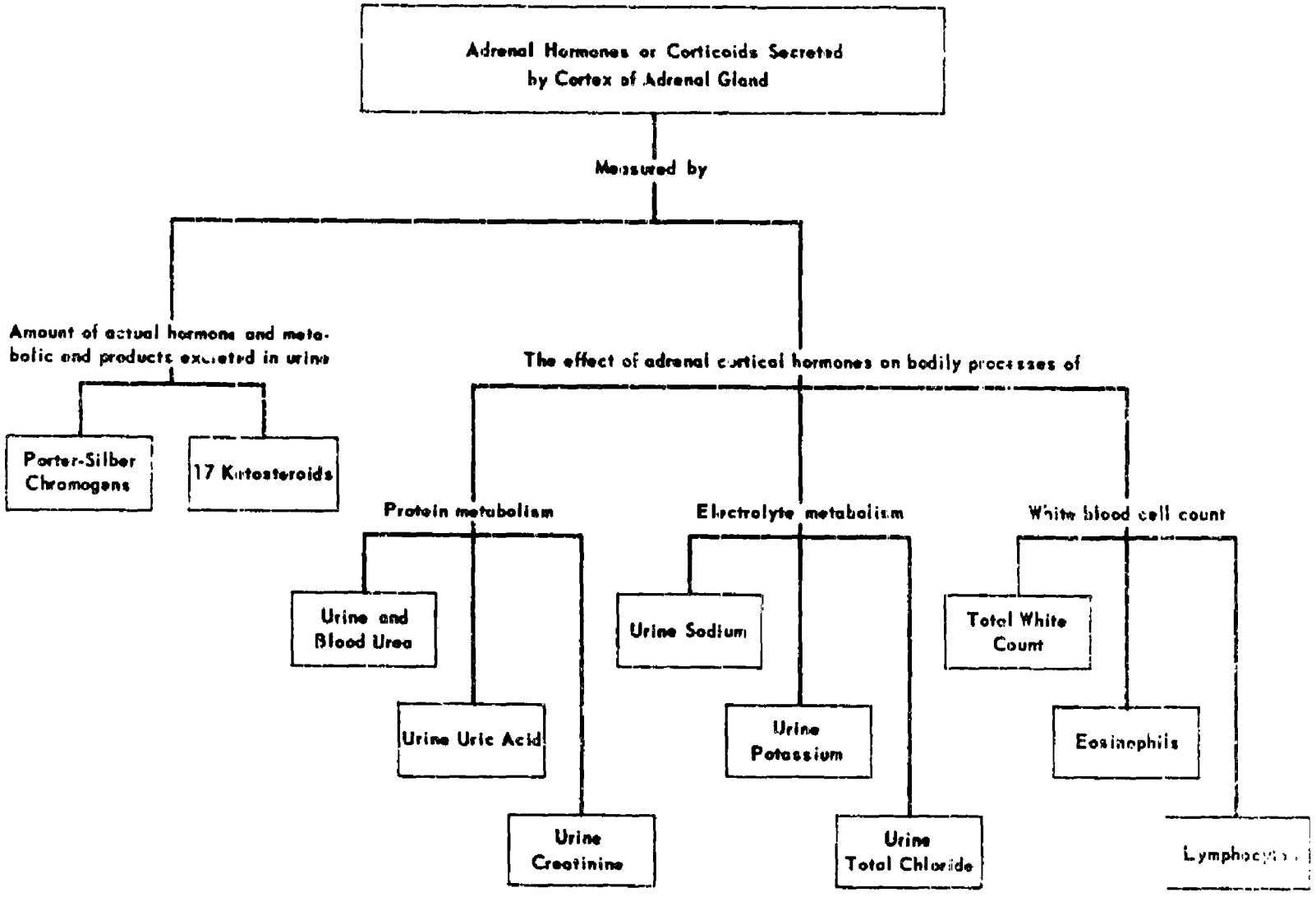


Fig. 2—Measures of Adrenal Cortical Function Used

reducing the initial increase there is a decreased excretion of these substances in the urine to normal or below normal levels.

(b) The measurement of the effect of the adrenal cortical hormones (corticoids) on body functions. Important to the body economy is the rate of protein metabolism (essential for growth, repair, and formation of body cells and as an energy source) and electrolyte metabolism (essential to the proper functioning of body cells through maintenance of electrical charges on cell membranes). The adrenal hormones markedly affect these processes and alter the rate at which they occur. With increased adrenal hormone secretion under stress these processes are changed, and the degree of this change can be recorded by measuring the by-products of these processes appearing in the blood and urine.

The by-product of protein metabolism is essentially nitrogen, which appears in the blood and urine in several forms. The nitrogen measurements in this study were urine and blood urea, urine uric acid, and creatinine. With stress these excretory products follow the same general pattern as P.S. chromogens and 17 KS, i.e., initially there is an increase indicating increased protein destruction (protein catabolism) and with continued stress there is a decrease to normal or below normal excretion.

The electrolyte balance which is largely dependent on concentration of salts in body fluids is indicated by the amount of these salts (sodium chloride, potassium chloride, and calcium chloride) in the blood and urine. In this study the effect of adrenal hormones on electrolytes was determined by measuring the amounts of sodium, potassium, and total chloride in the blood and urine. With stress, sodium and chloride are retained initially, i.e., excretion is decreased. With continuing stress, the excretion returns to normal or slightly above normal. Potassium, on the other hand, shows the reverse pattern: an initial increased excretion followed by an excretion at normal or a little below normal levels as stress continues. It is customary to express sodium and potassium output as a ratio of one to the other.

Stress also affects the white blood cell count, and, although the adrenal hormones probably play a part in this change, other factors are undoubtedly active. The white blood cell determinations made in Korea include the total white cell count as well as counts of specific cells: lymphocytes and eosinophils. The total white blood cell count usually increases with stress due to an increased production of new white cells (immature polymorphonuclear leucocytes). This increase in total cells is accompanied by a decrease in eosinophils and lymphocytes.*

A summary of the measures employed, their relation to adrenal function and the typical pattern of change with stress is shown in Fig. 2.

* In all, 43 measures were made on each of the groups and are reported in ORO-T-41(FEC). Some of these proved to be unreliable and others though reliable showed no effect of combat stress.

PROCEDURE AND RESULTS

The psychological and physiological measures described were administered to three groups of infantrymen in Korea. Two of the groups were engaged in active combat of varying intensity and length. The third group, which was not committed to combat, served as a control group for purposes of comparison.* To aid the reader in identifying the two groups committed to combat, they are identified by the type of action in which they were involved, i.e., Attacking Company and Defending Company. It is to be understood, however, that when the terms "Attacking Company," "Defending Company," or "Control Group" are used, they refer to the sample tested, not to the entire unit. In the following sections the groups and their responses to the measures are described in a general way. Detailed statistics and results of the analysis are presented in the appendices.

Control Group

The Control Group comprised 24 men selected at random from three rifle companies of the 15th Infantry Regiment, 3d Division, which had been assigned to a regimental blocking position immediately behind the MLR on the central front in Korea. They had been in these positions for 1 week, having previously been in corps reserve for 3 weeks. At the time of testing they were not engaged in combat, but they were called on for patrols. Their position was not one of complete safety, e.g., one subject was killed by a mine, and they were subjected to occasional artillery fire. Measurement was made twice on this group, the testing times (A and A') separated by 11 days as shown in Fig. 3.*

The physiological data of the Control Group compare very closely to that in laboratory work on normal subjects in the United States. The similarity is marked in spite of some expected differences in observations due to diet, time of collection, and general living conditions. It can therefore be concluded that soldiers in blocking positions some 200 yards behind the MLR, not in active combat, react normally on all the physiological measures used.

The psychological data are not absolute in that no norms suitable for comparative purposes are available. Since the administration and conditions of testing varied from established standards, the psychological data on this group serve as a reference baseline for the stress groups. There was a decided trend toward higher scores on the second administration of these tests (A'), undoubtedly due to the effect of practice.

*The Control Group was designated as Groups V and VI in ORO-T-41(FEC).

In early October the US forces planned a regimental size attack on the high ground in front of the US 7th Division sector. Both of the groups used by this team to assess the effects of combat stress were involved in this action.

Attacking Company

Able Company of the 31st Infantry Regiment, 7th Division, was the lead company in the initial attack on the Triangle Hill area north of Kumhwa. At 1700 13 October, Able Company was moved from the division reserve area to positions close to the MLR. They rested 3 to 4 hours, then moved out to the

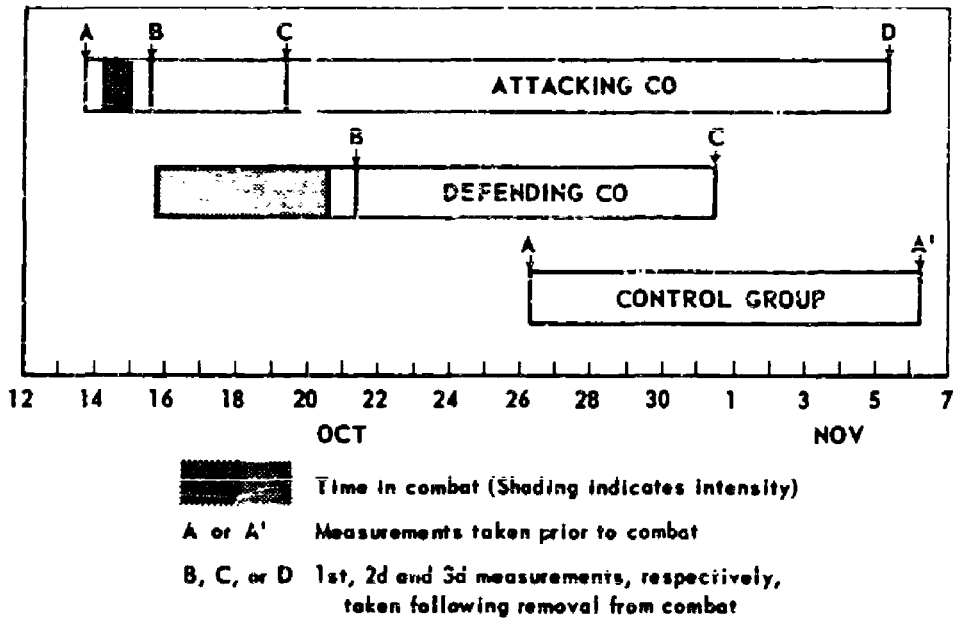
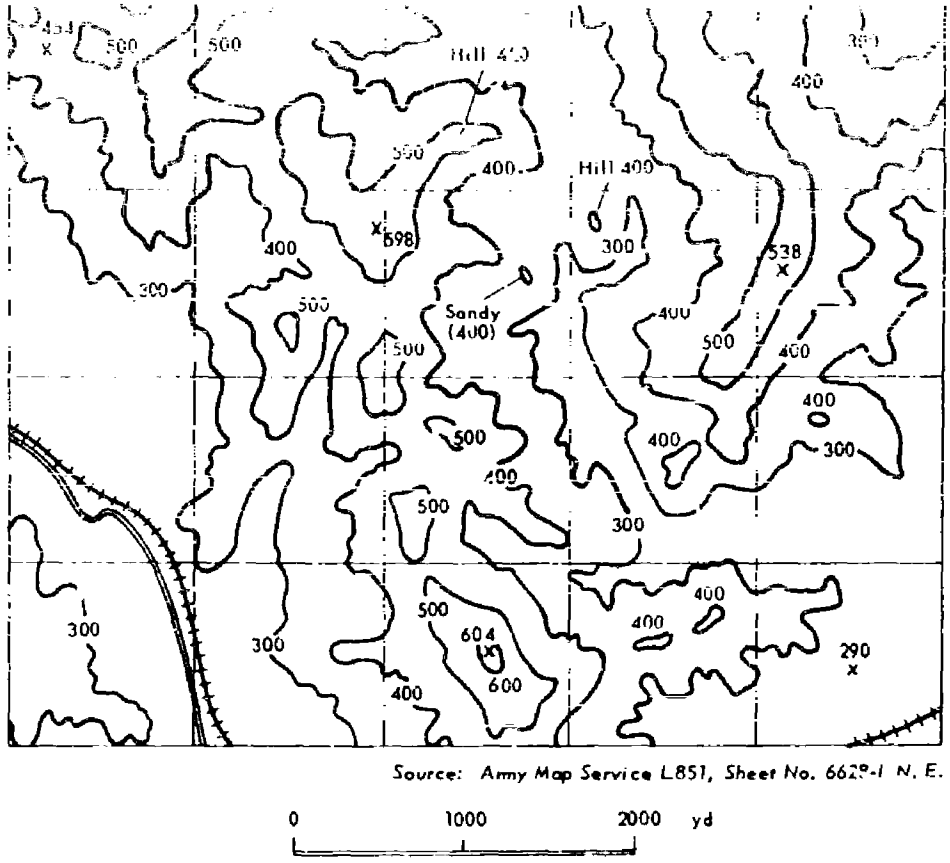


Fig. 3—Measurement Times of Groups Relative to Time in Combat
Period of testing: 13 Oct to ' Nov 52

MLR at 0430 on the 14th in preparation for the attack to begin at 0600. There was some delay in clearing a mine field, and so the unit did not pass through the MLR until 0615. Up until this point they had received no enemy fire. At 0730 they reached the base of the Triangle Hill Sniper Ridge complex where the company was split (see Fig. 4), the 3d Platoon going to the left, up the ridge to Sandy, and the 1st and 2d Platoons proceeding up the right ridge of Hill 400.

During this period they were subjected to artillery fire and suffered a few casualties. The 3d Platoon proceeded to move up toward Sandy, took out an enemy machine gun nest, and reached the peak. At this point they were hit by heavy fire from Hills 400 and 598. Most of the platoon withdrew from the peak and stayed in these more protected positions until portions of [illegible]er Company came up. They withdrew from Sandy at approximately 1[illegible] and proceeded up Hill 400 to help in the attack on that heavily fortified position.

The 1st and 2d Platoons advanced up the right ridge of Hill 400 under heavy fire from the fortified point on the ridge, from the peak, and from 598. At 0900 the 1st Platoon was within 35 yards of the enemy positions on top of Hill 400. There they were met by numerous grenades. At 1000 the 2d Platoon



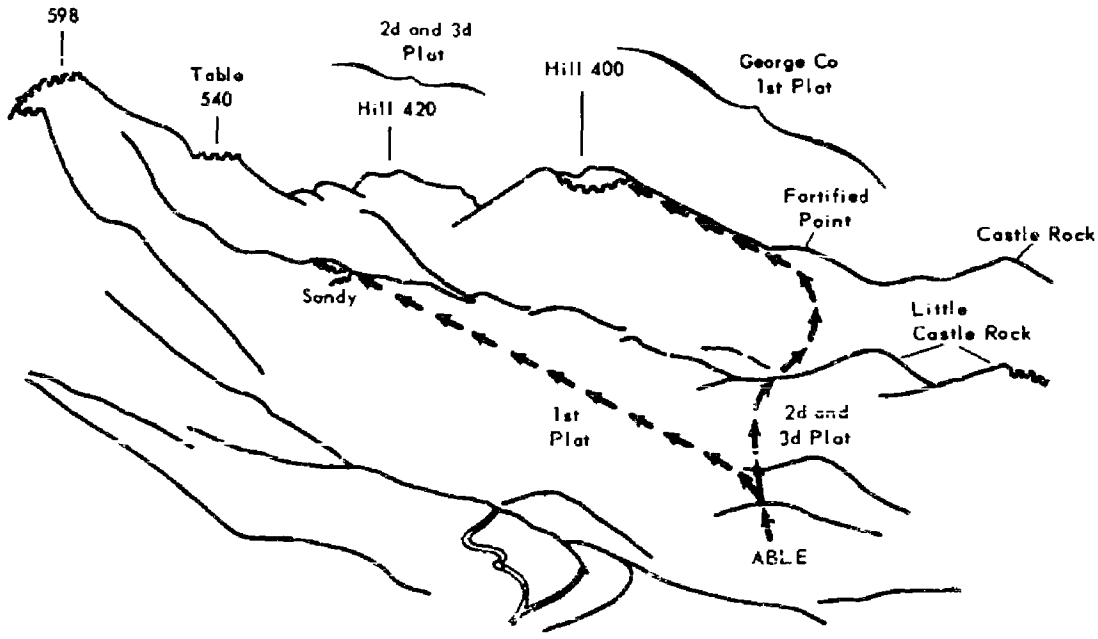


Fig. 4--Map of Able and George Company Area

joined the 1st. They remained dug in until 1100 when Baker Company moved through them to take the hill. By 1400 they had taken both peaks of Hill 400. At 1800 Able, Baker, and Charlie Companies moved up from Hill 400 toward 598, under heavy mortar and machine-gun fire. By 2000, the mortar fire had increased, and at this time the Chinese Communist Forces (CCF) mounted a counterattack. Under this pressure the companies were ordered to make a slow withdrawal. Able Company was back behind the MLR by 0200 on the 15th. On their return they ate C rations and slept. At 0700 they went to a collecting point where trucks took them to a blocking position. At 1100 they were returned by truck to the division reserve area, where at 1230 they were met by the research team.

The company was withdrawn from the attack after approximately 16 hours and was back at the MLR some 2 hours later, making a total of 18 hours of combat. They had been in severe combat, facing strong, well-prepared, and supported enemy positions. They had suffered 61 percent casualties,* and, at the time of testing following combat, the remaining 39 percent of the company appeared to be a very tired group who had been through a severe physical and emotional ordeal. Forty-eight hours after removal from combat they were moved to positions along a quiet sector of the MLR. Measures taken on this group include a precombat (A) sample on 24 individuals obtained some 2 hours after the final briefing for the attack and 12 hours before leaving the Line of Departure (LOD). The intention of this sample was to determine the condition of the men prior to combat and would include any effects due to anticipation. Time prohibited obtaining psychological data on the A sample. Some 12 hours after they were withdrawn from the fire fight, a B sample was obtained. There were only 5 of the original A sample available for testing at B since the remaining 19 had become casualties of one sort or another. Fifteen additional subjects were sampled from those returning in the company to increase the sample size to 20 for the poststress measures. The B measure was obtained as soon after combat as was possible in order that the effects of combat could be determined. To obtain data on the pattern of recovery, measures were taken on the same subjects 5 days (C) and 22 days (D) after the principal engagement.† The timing of these four measurements is shown graphically in Fig. 3.

The majority of the measures taken prior to combat (A) showed some effects of stress due to anticipation and/or the activity involved in preparation for the attack. However, the data are not markedly different from what is considered normal.

*The following breakdown of the casualties suffered by Able Company in this 18-hour action was obtained from Morning Reports.

Group	No. present for the action	Casualties					
		KIA	MIA	SWA	LWA	Total	%
Officers	5	—	—	2	3	5	100
Enlisted Men	165	20	6	18	54	98	59
Total	170	20	6	20	57	103	60.6

†The Attacking Company (Able) was designated Group III in ORO-T-41(FEC).

bat (B) demonstrate conclusively that adrenal responsiveness was increased and other physiological functions were significantly altered as a result of 18 hours of intense combat. An increase in production of corticoids is evidenced by the increased excretion of 17 KS and P.S. chromogens in the urine. An increase in output of urea, uric acid, and creatinine reflects a high nitrogen excretion as a result of increased protein catabolism. Evidence of a shift in the electrolyte balance is shown by a low sodium/potassium (Na/K) excretion ratio (due to high sodium retention) and a low total chloride output.

The total white blood cell count was decreased at B. Even though the expected increase in immature cells did occur, the total decrease is a result of the almost complete disappearance of mature cells. The eosinophils showed a drop immediately following combat; the lymphocytes, although decreased after combat, were not significantly lowered until 5 days following combat.

Five days after combat (C) most of the above measures had returned toward normal. Twenty-two days following combat (D) all the measures were within the range of normal values.

The foregoing description has been based on average or mean values. It should be pointed out, however, that in the Attacking Company there was a significantly wider range of individual values postcombat (B) on a majority of the measures when compared to the Control Group range. Even though there was an average change in a particular direction, some individuals showed an opposite or no change.

The psychological measures taken on the Attacking Company demonstrated no change that could be attributed to the effects of combat stress. It should not be concluded from this that there were no behavioral changes. The conclusion to be drawn is that any behavioral changes that occurred as a result of combat stress were not recorded by the measures used. The team felt there were definite changes in the men's behavior as a result of being in combat, but that these changes were not recorded because of one or more of the following factors: (a) the tests were not sensitive to the stress; (b) the time lag between stress and testing was too great, permitting recovery; (c) the subjects were motivated to exert compensatory effort at the time of testing; (d) the test conditions were not adequately standardized; and/or (e) practice effect masked any real deterioration of performance.

Defending Company

George Company of the 17th Infantry Regiment, 7th Division, occupied the same general combat area that Able Company had attacked, moving into the captured position 15 October, 1 ½ days after the initial attack (see Fig. 4). They held the combat position for 5 days before being relieved. During this period the military situation was relatively stable although enemy artillery was very active. The day prior to withdrawal they successfully repulsed a company-size counterattack. In addition they had withstood two additional enemy counterattacks in one of which their positions were temporarily overrun. In general the combat stress experienced by the Defending Company was more prolonged but less intense than that experienced by the Attacking Company. They suffered

approximately 17 percent casualties.* Twenty-four hours after being removed from combat they were moved into defensive positions in a relatively quiet sector of the MLR. No precombat data (A) were obtained on this company. Postcombat data were obtained on 13 men randomly selected from one platoon of the company some 12 hours (B) and 11 days (C) after the company was withdrawn from action.† These measurement times are shown in Fig. 3.

In contrast to the Attacking Company the data obtained on the Defending Company demonstrate conclusively that as a result of 5 days of less intense combat adrenal responsiveness was decreased, and in addition other of the physiological measures showed a different pattern of response from that of the Attacking Company. The decreased excretion of 17 KS and P.S. chromogens in the Defending Company following 5 days in combat (B) shows that there was a decreased production of hormones by the adrenal cortex. This decrease in adrenal hormones was accompanied by normal protein metabolism and a shift in electrolyte balance opposite to the shift in the Attacking Company, i.e., the Defending Company showed a high potassium retention and approximately normal sodium excretion resulting in a high Na/K ratio. The total chloride excretion was significantly lower than the Control Group.

TABLE 1

SUMMARY OF POSTSTRESS (B) DATA, ATTACKING AND DEFENDING COMPANIES

Measure	Attacking Co	Defending Co
P.S. chromogens	High	Low
17 KS	High	Low
Urine urea	High	Normal
Urine uric acid	High	Normal
Urine creatinine	High	Normal
Lymphocytes	Normal	Low
Urine sodium/potassium	Low	High
Total white count	Low	Normal
Eosinophils	Low	Normal
Urine total chloride	Low	Low

The total white blood cell count in the Defending Company immediately after combat (B) is also different from that of the Attacking Company, in that there was not as great a disappearance of mature cells, and the increased production of immature cells compensated for this, resulting in the maintenance of

*The following breakdown of casualties suffered by George Company in the 5-day defense was obtained from Morning Reports.

Group	No. present for the action	Casualties					
		KIA	MIA	SWA	LWA	Total	%
Officers	7	1	—	—	1	2	29
Enlisted Men	151	5	—	5	14	24	16
Total	158	6	—	5	15	26	16.5

†The Defending Company (George) was designated as Group IV in ORO-T-41(FEC).

a total white count in the normal range. The eosinophils were in the normal range, and lymphocytes were significantly lowered.

The most notable changes in the blood count in the Defending Company were recorded 11 days after leaving combat (C). At that time there was a significant decrease in total white cells and eosinophils. Some of the other measures had returned to normal levels. The remaining ones, although approaching normal, had not yet reached normal levels.

Again in contrast to the Attacking Company the range of individual values on the majority of measures was quite narrow, being the same or less than the Control Group and significantly less than the Attacking Company. In other words the response of individuals in the Defending Company was much more uniform than in the Attacking Company.

The Defending Company showed no demonstrable changes on the psychological measures which could be attributed to the effects of combat.

Table 1 presents a summary comparision of the poststress (B) response of the Attacking and Defending Companies on the physiological measures and emphasizes the markedly different response of the two groups on each of the measures. With the exception of the P.S. chromogens (which were not tested statistically) high or low indicates the direction of statistically significant differences from controls.

INTERPRETATION

The basic data have shown that the average physiological response of soldiers in an intense, short-term battle (Attacking Company) is different from the response of those in less intense combat for a longer period of time (Defending Company). Further there is evidence that the responses of individuals are not uniform in the intense situation (Attacking Company), but are quite consistent in longer, less intense combat (Defending Company). The data also show that the length of time taken for the physiological processes to return to normal differs for the two groups. Each of these problem areas—group differences, individual differences, and recovery times—will be discussed in light of its relation to military effectiveness.

Group Differences

In the Attacking Company all the physiological indices indicate that the adrenal cortex was active; i.e., that it was being stimulated by the stress encountered in the short, intense battle and was secreting a larger than normal amount of adrenal hormones. Accompanying this there was protein catabolism and a shift in the electrolyte balance.

In the Defending Company the converse is true. The measures indicate that the adrenal was not responding to stress, that it was secreting a lower than normal amount of hormones, that there was no protein catabolism, and that there was an opposite shift in the electrolyte balance. Thus there appears to be a stimulation of adrenal cortical function in short-term, intense combat, and a dulling or lack of stimulation of the adrenal cortical function in the long-term, less intense stress situation.

ACTH from the pituitary gland stimulates the adrenal cortex to produce hormones. The injection of ACTH likewise stimulates the adrenal gland, and measurement of the resultant excretory by-products provides an indication of the activity of the adrenal cortex. If the adrenal gland is hypoactive and therefore incapable of producing hormones, injection of ACTH would cause little or no change in the by-products excreted. On the other hand, if the adrenal is normal or hyperactive, ACTH will cause an increased secretion of hormones with their concomitant effects. ACTH was injected in several subjects of each group, following the completion of the other tests, to stimulate the adrenal gland to its utmost capacity. The response was measured by determining the excretory by-products (P.S. chromogens, 17 KS, Na/K ratio) in the urine collected over a 12-hour period.

The ACTH data support the distinction between the two groups and establish the fact that in the Attacking Company the adrenal was still capable of producing increased amounts of corticoids, whereas in the Defending Company the adrenal gland was incapable of producing stress-protective hormones.

Selye has, through a detailed analysis of stress literature, traced the course of various physiological responses during stress.[5] Generalized curves for several of these physiological responses are shown in Fig. 5. These responses follow

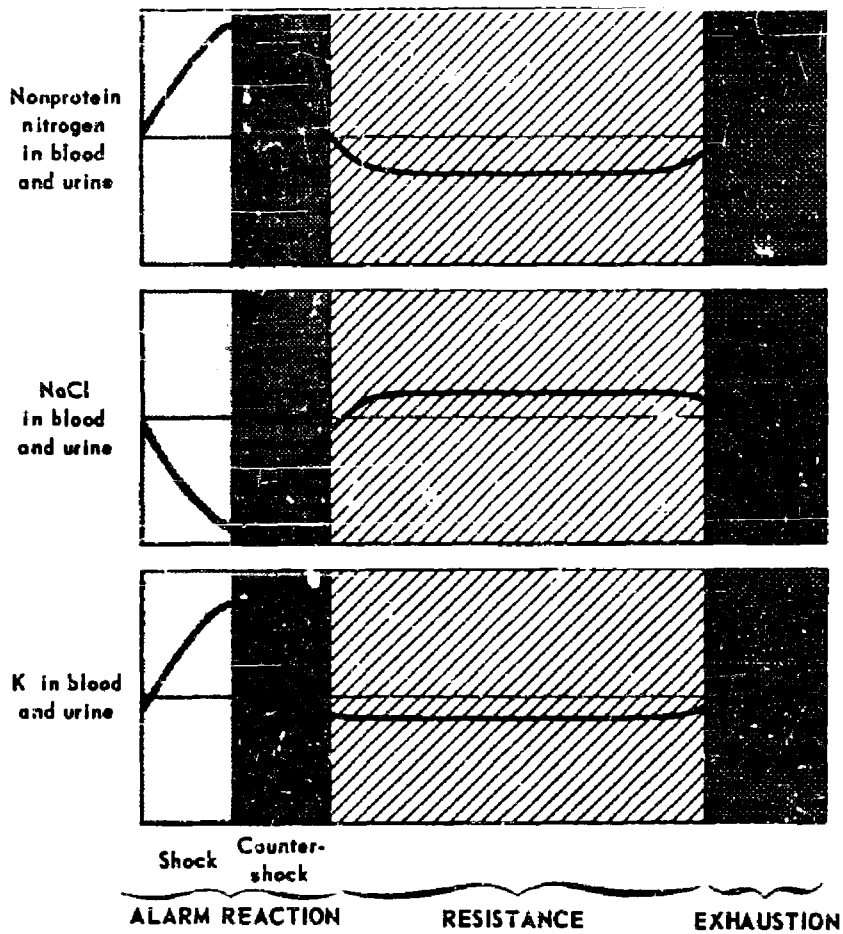


Fig. 5—Generalized Stress Curves[5(278-79)]

a rather characteristic pattern divided into alarm, resistance, and exhaustion phases. In general, when an organism is stressed, there is an initial period of marked deviation from normal called the "alarm reaction," followed by a period when the organism has adapted or developed resistance, and finally, if the stress is sufficiently prolonged, the state of exhaustion and eventual death is reached. The characteristic physiological manifestations of the alarm stage (protein catabolism, increased 17 KS excretion, sodium retention, potassium excretion) are no longer evident or are actually reversed during the stage of resistance.

From the poststress data presented in this study, it is evident that, although the responses of both groups can be considered normal stress responses, the Attacking Company was in the initial or alarm phase of this stress reaction,

whereas the Defending Company had passed through this stage and reached the second or resistance phase. The data on the Defending Company are unique, since they are the first record of humans reaching this resistance phase as a result of emotional or physical stress not involving trauma such as surgery or fractures. From this it can be concluded that combat has marked physiological effects.

The apparent consequences with regard to capability for withstanding added stress are different for the Attacking Company, which was in the alarm phase, than for the Defending Company, which was in the resistance stage. Selye states that during the alarm phase the organism's resistance to the stress to which it is being currently exposed is lowered at first and then begins to rise, so that when the organism reaches the stage of resistance it is maximally adapted physiologically and is more resistant to the stress. However, he further proposes that the organism's resistance to stresses other than the one to which it is currently exposed is also lowered initially, rises later in the alarm phase to slightly above normal, and then drops sharply to below normal on entering the resistance stage.[5(55)]

Therefore the pattern of the Attacking Company is in favor of the individual, especially with respect to the possibility of an added insult to the organism such as physical wound since it is in the late stage of the alarm phase. In this group the subjects, for the most part, have quite responsive adrenals. The organism is prepared to handle the result of physical wounds, such as loss of blood and the need to maintain electrolyte balance. However, they have not yet adapted to the stress to which they are exposed (combat) and therefore are less resistant to it. In the Defending Company the individual's general physiological condition indicates that with an added stress, such as a physical wound, the adrenal may be unable to accomplish its role of protecting the organism. The adrenal seems to be nonresponsive with regard to production of corticoids. However, following Selye's hypothesis, the Defending Company has adapted maximally to the combat stress to which they have been subjected and are therefore more resistant to it.

Individual Differences

The team agreed that all the individuals studied were subjected to severe stress, and hence similar degrees of physiological change were expected. In the Attacking Company every man was up against intense enemy fire, and all went through a similar physical exertion. With 61 percent casualties it is certain that each individual saw others severely wounded or killed and thought it likely that he might also become a casualty. In spite of this similarity of experience, an exceptionally wide range of physiological response was observed. Conversely in the Defending Company there was a narrow range of physiological response. Every member of the Defending Company was undoubtedly subjected to a similar degree of stress comparable to the stress encountered by the Attacking Company although it was more prolonged but less intense.

The observation of individual differences in response to stress is common, and it has been the general practice to attribute these differences in response to a difference in the meaning of the situation to the individual. If meaning were responsible for individual differences in this study, wide variation in response would be observed in both the Attacking and Defending Companies. Since there was wide variation in the Attacking Company and a very restricted range in the Defending Company, differences in meaning cannot reconcile such an apparent discrepancy.

This discrepancy can be accounted for, however, by applying a general pattern of physi logical response to stress which is similar for each individual, and through which each individual proceeds at a different rate. Such a generalized pattern has been recorded by various investigators and in general it follows Selye's previously described triphasic curve. Similar response patterns, with different rates for each individual, are graphically presented in Fig. 6. In this

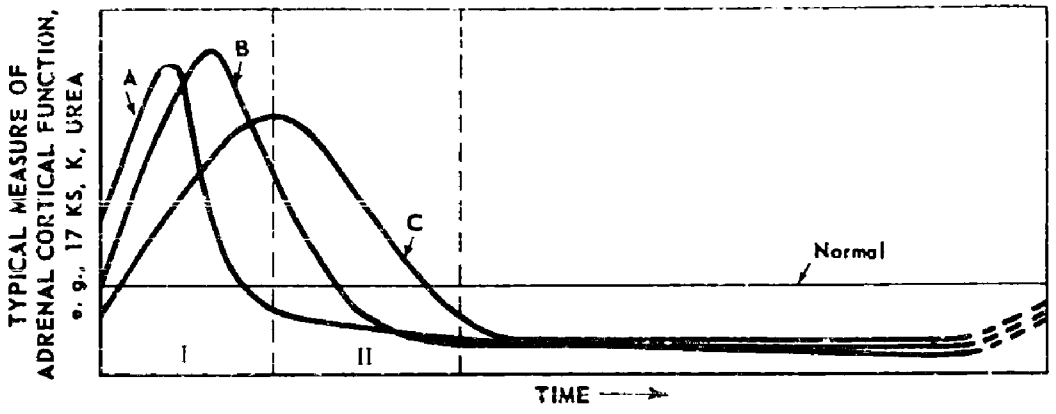


Fig. 6—Variation in Rate of Physiological Pattern Shown by Three Individuals, A, B, and C, in Stress Patterns I and II

figure the response of a typical physiological measure to prolonged stress is plotted against time. Assume that stress starts acting at time 0 and continues for an indefinite period. The reactions of three hypothetical subjects A, B, and C are shown by the three curves. Individual A, who normally has a high output, may respond very rapidly at the onset of stress and proceed through the pattern much more rapidly than Individual C whose initial output is lower and whose rate of change may be slower. It can be seen from this graph that if this response were measured at Time I there would be a wide range in the values obtained at that time; Individual C is in the alarm stage and shows a high response; Individual A has entered the resistance phase and his response is below normal, and Individual B is more advanced in the pattern than A but has not reached the stage that C is in. Values obtained at Time II would be very similar since all individuals would have passed into the second phase of the stress pattern.

Applying this reasoning to the physiological data obtained in Korea, the apparent discrepancy in variability between the Attacking and Defending Companies can be explained. The 17 KS data in the Attacking Company, for example, shows a high mean poststress value, but the individual measurements range from considerably below normal to 5 times above the normal value. The poststress measurement of the Attacking Company was taken close to Time I and includes individuals in both the alarm and resistance phases. The 17 KS data for the Defending Company have a below-normal mean and a restricted range, with all individuals being at normal or below normal. The data indicate that the Defending Company, after stress, was measured in the region of Time II and that all individuals were near or in the resistance stage.

There is further evidence to support this. If the assumption is correct and those individuals in the Attacking Company who were low postcombat 17 KS excreters have in fact entered the resistance phase, then the mean response

the Defending Company since the Defending Company has been previously identified as being in the resistance phase.

Seven of the eight lowest 17 KS excreters at B in the Attacking Company show an increased 17 KS excretion at C. A comparison of these eight lowest excreters with the Defending Company shows marked agreement of other physiological data.

In the section "Group Differences" it was pointed out that the Defending Company was suffering from a depletion of certain stress-protective hormones and that this condition is physiologically undesirable if the individuals are subjected to other stresses. It is evident that after 18 hours of intense combat 44 percent of the Attacking Company had already reached this condition whereas the remaining 56 percent had not yet reached the stage of resistance. Defending Company data indicate that a much larger proportion were in the resistance phase.

Recovery Time

Important to the tactical employment of combat units is the knowledge of both deterioration as combat continues and the time taken to recover sufficiently for further combat. From the data obtained in Korea, conclusions have been drawn regarding deterioration in combat. In addition these data permit conclusions to be drawn pertaining to the approximate rates of recovery for the Attacking and Defending Companies.

Referring again to Fig. 3, the Attacking Company was measured 3 times following removal from combat: at 12 hours, 5 days, and 22 days; the Defending Company was measured twice, at 12 hours and again 11 days after combat. In the Attacking Company the electrolyte, nitrogen, and 17 KS measured had returned to nearly normal levels when measured 5 days after combat. The blood counts were approaching normal levels although they cannot be considered normal. On the other hand the Defending Company after 11 days out of combat was still low on 17 KS and blood counts had dropped even further. Electrolytes and nitrogen were in the normal range. It can be concluded from these observations that exposure to even 18 hours of intense combat requires several days for physiological recovery. Assuming a linear relation between time following combat and increments of physiological change toward normal, approximate recovery times can be stated for each of the groups studied. The Attacking Company recovered in approximately 6 days, certainly not less than 4 and not more than 8 days. The Defending Company recovered in approximately 13 days, certainly not less than 10 or more than 16 days.

The Attacking Company spent 18 hours or 0.75 day in combat, the Defending Company 5 days in combat. Thus for each day spent in active combat, the Attacking Company required approximately 8 days (6 days divided by 0.75) to recover. The Defending Company required approximately 2 to 3 days (13 days divided by 5 days) to recover for each day spent in combat. This may be interpreted to mean that the relative cost in recovery time was much greater for the Attacking than for the Defending Company.

This difference in the relative cost of combat, 8 days for the Attacking Company and 2 to 3 days for the Defending Company, leads to the following conclusion: the cost of combat stress in terms of physiological recovery time

increases during the initial stages of adjustment and/or increases with increasing intensity.

It has been determined from the data that the Attacking Company, on the average, was still in the process of adjusting to the stress. The Defending Company had reached the stage of resistance where adaptation to the stress to which they were exposed was maximum. Since the time taken to recover per day in combat was less for the Defending Company than for the Attacking Company, it is logical to conclude that the cost of adapting in terms of recovery time is higher than the cost of maintaining maximal adaptation.

The Attacking Company, however, was exposed to intense combat, the Defending Company to combat of less intensity. This difference of relative cost for the two groups may demonstrate a marked effect of increasing intensity. It is commonly thought that one of the primary effects of increased intensity of stress is to increase the rate of physiological change. Thus with an increase in the intensity of combat a given physiological state (a state requiring a given recovery time) would be reached after a shorter time in combat. Therefore time taken to recover per day in combat would be greater.

The observations in this study and the experimentation of others indicate that both intensity and the stage of adaptation are operative.

IMPLICATIONS

Every military unit performs combat missions at a cost. The sum of the tactical activities directed toward accomplishment of the tactical mission is the useful output of the unit. The immediate specific output of a small combat unit is typically fire and movement. This output may be measured in such terms as rate and duration of fire and rate and range of maneuver. The sum of the expenditures required for accomplisnment of a tactical mission is the cost of the operation. The specific costs are typically cost of ammunition and supplies expended, equipment damaged, and the casualties sustained, which may be measured by casualty replacement effort and dollar cost of weapons and ammunition replacement. The casualty replacement effort includes the men used for replacement as well as all the money, time, and facilities required to rectify functional deterioration incurred in combat. Such functional deterioration, as used in this report, includes the temporary functional impairment induced by combat, as well as militarily recognized physical casualties.

Efficiency of a Unit

The efficiency of a military unit in the performance of a tactical mission can be defined as useful output divided by cost. Some of the factors which comprise output and cost are directly affected by stress of combat. Although the effect of stress on troops is primarily in the category of a temporary functional impairment not recognized as a casualty, it also immediately alters the combat output (fire and movement).

The efficiency defined above as useful output divided by cost demonstrates a means which assists in determining the best use of units in combat. It is not yet clear how, even for a small tactical mission, the total useful output and total cost may be computed to yield the efficiency for the operation. It may be possible, however, to compare partial efficiencies for similar operations. By partial efficiency of an operation is meant the ratio of portions of the useful output to corresponding portions of the cost. Such partial efficiencies will be significant only insofar as the effect of the remaining efficiencies is fixed or known.

Relation of Efficiency to Combat Stress

The conclusions stated previously that relative cost in terms of recovery is a function of intensity and/or stage of adjustment to combat stress suggest the following hypothesis relative to efficiency and stress: the partial efficiency which is a function of stress decreases as intensity increases; and at any given intensity, efficiency is least while the unit is adapting to combat stress. This hypothesized relation is represented by the efficiency curve (solid line) shown

in Fig. 7a. The curve represents the relation of time in active combat to physiological recovery time for a unit engaged in combat of relatively low intensity. For any given intensity the first part of the curve (along the vertical axis to line A) represents the initial or alarm phase of the stress reaction, which is the least efficient or the most costly in terms of number of days taken to recover

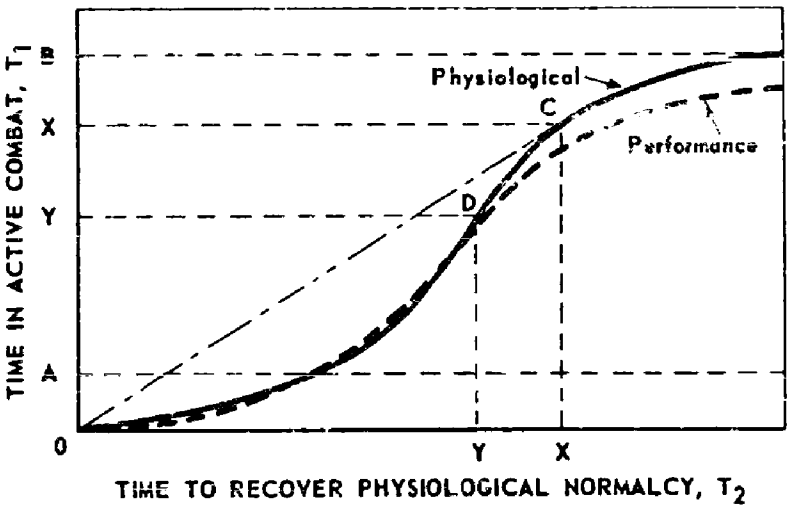


Fig. 7a—Recovery Time vs Time in Combat (Hypothetical)

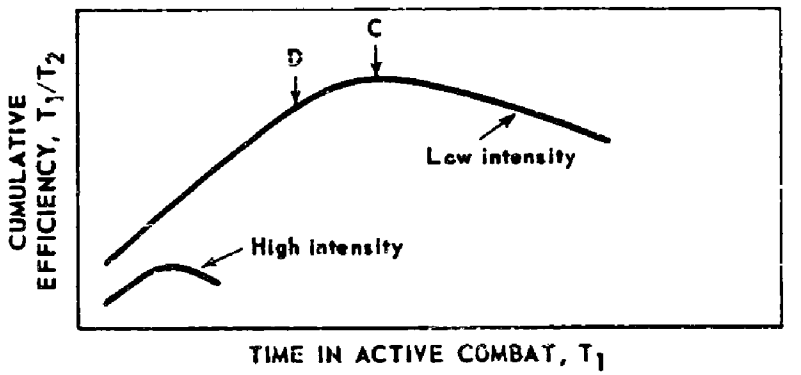


Fig. 7b—Cumulative Efficiency Curves for High- and Low-Intensity Combat

per day in combat. The period of resistance follows (area between line A and line B). During this period adaptation is maximum and efficiency increases to maximum at Point C, after which a gradual fall-off in efficiency occurs as the stage of exhaustion is approached. To exhibit the variation of this efficiency with combat time, Fig. 7b shows the cumulative efficiency itself: T_1/T_2 plotted against combat time T_1. The effect of high intensity on efficiency can be seen from the lower curve of Fig. 7b. With high intensity exhaustion is reached sooner and the efficiency is much lower.

The curves in Figs. 7a and 7b represent only one output and one cost. The complete relation of stress to efficiency must consider other outputs and costs. For instance, the solid curve in Fig. 7a does not take into consideration changes in performance with combat, which is obviously a prime consideration. From

... initial decrease in performance, followed by overcompensation, resulting in improvement of performance during the early stages. This overcompensation gradually disappears as adaptation becomes complete. Ultimately performance decreases with the continuation of stress for long periods. For low-intensity combat this might result in the curve shown in dotted lines in Fig. 7a, where the output now includes the effect of performance changes incurred by stress. This increases efficiency in the early stages and decreases it in the later stages. The behavior patterns that have been observed under extreme stress are variable, ranging from completely ineffective behavior, such as immobility, to highly effective behavior such as feats of strength which could not be repeated under normal conditions. This apparently is an individual phenomenon, and it is difficult to hypothesize a curve for average behavior.

The cumulative efficiency of a combat unit is highest for an action which ceases at the time indicated by the maximum value of T_1/T_2. This point (C) is shown graphically in Fig. 7a by the steepest straight line (line OC) that can be drawn from the origin tangent to the curve. The point of highest instantaneous efficiency (D) occurs where the slope of the curve is steepest and as such is lower on the curve than the point of highest cumulative efficiency. The point of maximum cumulative efficiency (C) is the time to withdraw units from combat. Further engagement of that unit proceeds at higher cost in recovery time or combat time.

These curves are hypothetical; the general trends are based on data, but the absolute values are unknown. With sufficient data to construct actual curves, their application to a combat situation is apparent. If the commander anticipates an engagement of the duration of approximately twice the optimum combat time for maximum efficiency (2X) and if he has available several combat units and no other impending missions, then it is clear that his task can be most efficiently accomplished by successively committing two units, each for a period of $T_1 = X$. If, instead, the commander choses to commit only one unit for the entire duration, this unit will be operating at reduced efficiency approaching exhaustion during the later half of the engagement. On the other hand, should the commander elect to commit successively three or more units for periods of time T_1 less than X, these units will be operating largely during their period of adaptation at low efficiency.

With the information provided by actual curves, such as those which have been hypothesized but which are based on complete output and cost data, the relative efficiencies of units as a function of stress could be determined. To provide such curves, the course of performance changes with stress must be determined since the ultimate solution of the military problem defined here depends on knowledge of the decrement in performance of such tasks as delivering effective fire, speed of movement, and appropriate response to commands. Future work on the problem of stress effects of combat will be primarily directed toward determining effects on performance.

The data provided in this study plus data which will be obtained on performance, casualty rates, and physiological condition as a function of length of combat must all be integrated in order to compute the relative efficiencies of units. This information will be of considerable assistance to a commander in making such decisions as how long a company should remain in combat, which of several units is in best condition to perform in further combat, or when a unit can be used again.

Appendix A

PHYSIOLOGICAL MEASUREMENT

CONTENTS

TABLES (CONTINUED) Page

INTRODUCTION

When the body is subjected to stress there is an attempt to maintain equilibrium or homeostasis. Cannon explored the maintenance of homeostasis in the face of stress and directed attention to the role of adrenalin and the autonomic nervous system in bringing about the adjustments on which homeostasis depends.[6] His emphasis was mainly on respiration, the circulatory and gastrointestinal systems, and body temperature.

Recently the emphasis has been on the role of the adrenal-pituitary system in stress. Here the work has been directed toward biochemical measurements related to adrenal cortical function. When the organism is exposed to a variety of nonspecific stresser agents there is not only a discharge of adrenalin, but also excretion of adrenal cortical hormones. The response is apparently nonspecific, i.e., any stress seems to be met by an increased discharge of adrenal cortical hormones.

It is quite certain that the stress condition causes the pituitary gland to secrete adrenocorticotrophic hormone (ACTH). This in turn activates the adrenal cortex, which releases principally corticosterone (Compound B) and 17 hydroxycorticosterone (Compound F) which have far-reaching metabolic repercussions in the body. There is normally a 2 to 1 ratio of F to B in the human peripheral blood.[7]

There are considerable experimental and clinical data indicating the involvement of the adrenal cortex in a wide variety of stress situations.[5] Stresses such as cold, fatigue, infections, burns, etc., affect large portions of the body and have a description and response specific for the stressing agent, and in addition there is a nonspecific response which involves the function of the adrenal cortex. The organism cannot withstand or make adjustments to stress without the proper functioning of this gland.[1(111)]

The measurements of the adrenal cortical function are in general of two kinds: (a) the amount of actual hormones secreted or the metabolic end products of the hormones excreted in the urine [e.g., Porter-Silber (P.S.) chromogens and 17 ketosteroids (KS)], and (b) the measurement of the effect of the hormones on tissues and organs (e.g., white bloods cells, cholesterol, protein metabolism, and electrolyte balance).

Another way of looking at the pituitary-adrenal function under stress is to study the basic physiological processes affected by adrenal hormones.[7] It appears that the adrenocortical hormones

> ...support the capacity of the tissues to attain peak rates of metabolic processes when such are required to sustain homeostasis. Thus the adrenocortical hormones support the capacity of the kidney either to retain sodium and water when retention is needed,

or to excrete an excess. These hormones support the conservation of carbohydrate during resting as well as support its peak utilization for work when high energy output is required. The same hormones will support either growth or the rapid mobilization of tissues when such is needed during stress.[8(34)]

No metabolic process is known to be abolished by adrenocortical insufficiency, and no new process is known to be created when these hormones are administered in excess. It is supposed that these and other hormones enter into body economy by affecting the rates of metabolic processes.[8(23)]

It is established that the secretory activity of the adrenal cortex is greatly increased during stress although the amounts of adrenocortical hormones secreted are still undetermined. It is known through animal experiments that the adrenal cortex can secrete sufficient hormones to cause metabolic upsets, tissue pathology, and death.[8(29)]

In brief and in the final analysis it is the function of the pituitary-adrenocortical axis to secrete hormones during stress. These hormones permit the organism to resist, adapt, survive, and return to a normal state following a stress situation.[1(37)]

Pituitary-adrenal function was therefore the major target of the measurements selected for study. The description of the individual measures, their administration, and results are given in ORO-T-41 (FEC).[2]

By the nature of this project there were many uncontrollable and unavoidable variables. It is a well-known fact that many of the physiological processes vary from time to time during the day (diurnal rhythm) and are affected by diet and water intake. A concerted effort was made to obtain successive samples at roughly the same times and to become acquainted with the food and water intake of the subjects. These have been studied with the result that it is felt that none of the striking results could be due to diet or any of the other uncontrolled factors.

MEASUREMENT RESULTS

Eosinophils

Eosinophils compose approximately 3 percent of the white blood cells. They are subject to a diurnal variation and a wide normal range, approximately 75 to 350 cells per cubic millimeter (cu mm). A decrease in the level of circulating eosinophils in the blood is an index of stress although falls as large as 40 percent have been observed without apparent cause.[9] It seems evident that a reduction in the level of circulating eosinophils (eosinopenia) following stress may be a result of mechanisms other than an increased adrenal corticosteroid secretion since eosinopenia has been observed in the absence of the adrenals.[10] However, eosinopenia is accepted as a clear indication of stress whether mediated through the adrenal cortex or the autonomic system. The level of circulating eosinophils changes quickly, dropping soon after stress begins but returning to normal within a short time. It is postulated that such a phenomenon occurs because of the large involvement of the autonomic nervous system. In addition, although there is a drop in the level of eosinophils following injection of ACTH, it is a slow response.

Eosinopenia has been observed following operations,[5(406)] upon exposure to cold,[5(408)] in medical students just prior to examination when emotional tension is high,[11] and in subjects following 2 hours of working multiplication problems.[12] It would appear that emotional stress is stronger than physical stress as an activator of eosinopenia, for little or no decrease in eosinophil level was observed where subjects were worked to exhaustion on a treadmill.[13]

The count of eosinophils in blood samples withdrawn 1 hour after completion of the 500-mile Indianapolis Speedway Race (1950), or withdrawal of the driver from the contest, showed better than 60 percent decrease in all subjects when compared to the eosinophil counts taken a week prior to the race.[14]

TABLE A1

EOSINOPHIL COUNT, CONTROL GROUP[a]

(per cu mm)

Mean A measure	Mean A' measure	Mean diff.	t	df.	P
207.6	197.9	9.7[b]	0.57	16	NS

[a] The measurements A and A' on the Control Group were separated by 11 days. The Group is described in the section "Procedure and Results" in the main text.

[b] Means are based on individuals (N = 18) on whom both A and A' are available. One individual was eliminated (leaving N = 17) for having an eosinophil count of over 600 per cu mm. (890 on A and 475 on A'.) This is an arbitrary cutoff point. Eosinophil counts are commonly used as an index of certain infections. Therefore any value above 600 is assumed to indicate a pathological condition of the subject and was not included with the data. This procedure was followed in all the groups, with an indication in the footnotes as to the ones dropped. In all instances where the measure was over 600 cu mm the individual's data were dropped completely. A count of 700 eosinophils per cu mm was considered gross eosinophilia by the British in a study on Commonwealth soldiers in Korea and the values were dropped from the data.[16]

A study of the Harvard varsity and combination crews prior to and after the Harvard-Yale 4-mile boat race showed that, in general, the eosinophil level of the varsity crew after a "spectacular and frightening finish" had dropped 90 percent below prerace level. The same situation did not hold for the combination crew, which easily won its race; there was no eosinophil drop but instead a slight mean increase over prerace values. "Emotional stress, either in itself...or in combination with muscular activity...represents a maximal stimulus for the stress mechanism studied, whereas this does not seem true of muscular activity alone."[15]

As shown in Table A1, the mean eosinophil counts of the Control Group were not significantly different from A to A'. There was a correlation of $r = .84$ (significant at the 1 percent level) from A and A'. This indicates that the Control Group, even though living on the MLR, was not subjected to conditions which resulted in a systematic variation in eosinophils, and that eosinophils are a reliable measure.

As shown in Table A2, the Attacking Company showed an eosinopenia after 16 hours of active combat. The mean at B is significantly lower than the Control Group mean and lower than the Attacking Company mean at D. The means show

5 days after leaving the assault area (C) the group eosinophil counts had increased, but not significantly, and showed a significant increase when measured 22 days (D) after leaving the assault area. The recovery from combat as measured by the level of circulating eosinophils was therefore in terms of days, in this case a week, rather than hours.

The precombat data (A) were taken on 24 subjects some 2 hours after the final briefing for the attack and 12 hours before leaving the Line of Departure (LOD). The intention of this sample was to determine the condition of the men prior to combat and would include any effects due to anticipation. From Table A2 it is evident that the eosinophil count prior to combat was normal

TABLE A2

EOSINOPHIL COUNT, ATTACKING COMPANY[a]

(per cu mm)

Means compared[b]	Mean diff.	t	df.	P
Attacking Co A (215) vs Control Grp A (235)	20	0.48	40	NS
Attacking Co B (149) vs Attacking Co C (176)	27[c]	1.96	17	.10 > .05
Attacking Co B (148) vs Attacking Co D (227)	79[c]	3.54	14	< .01
Attacking Co C (165) vs Attacking Co D (226)	61[c]	3.50	15	< .01
Attacking Co B (149) vs Control Grp A (235)[d]	86	2.30	39	< .05

[a]Measurements were taken on the Attacking Co before combat (A), and 14 hours (B), 5 days (C), and 22 days (D) following combat. The Attacking Co is described in the section "Procedure and Results" in the main text.

[b]Numbers in parentheses in all tables refer to means compared.

[c]Means are based on individuals on whom paired data were obtained. This does not apply where different groups are compared. On the basis of 600 eosinophils per cu mm cutoff point, one individual was eliminated (685 on B, 810 on C, 770 on D).

[d]On the basis of 600 eosinophils per cu mm cutoff point, one individual was eliminated (390).

since it did not differ significantly from the mean of the Control Group. (An analysis of the lymphocytes and total white blood cell count shows that they did not differ from normal precombat data either.)

Only 5 of the 24 men sampled at A were available for further testing following combat, and although the mean was not significantly different from the Control Group there are some indications that the data were modified somewhat by anticipation of combat. This is confirmed by lymphocytes and white blood cells (see following sections). It was for these reasons that the data are compared to the Control Group and not to other measures taken on the Attacking Company.

The eosinophil count in the Defending Company (Table A3) shows a different response from that of the Attacking Company. There was no eosinopenia in this group when measured 12 hours following combat. However, 11 days after leaving the assault area the mean was significantly lower than the Control Group mean.

Taken as a group there was an eosinopenia in the Attacking Company; however, it is interesting to look at the response of the individuals. Here was a situation where the stress was severe and in which the meaning might appear

to have been uniform since there was a universal danger. Even so, there were three individuals who showed a higher eosinophil count on the stress (B) than on the recovery (D) measure and a fourth whose recovery count was less than 1 standard deviation increase over the stress measure. The fact that these four individuals deviated from the general stress pattern may result from measurement errors, variations in diet, and level of activity, or they may reflect a true, physiologically different response (rate or time of response) to the stress, thereby resembling the response of the Defending Company.

TABLE A3

EOSINOPHIL COUNT, DEFENDING COMPANY[a]

(per cu mm)

Means compared	Mean diff.	t	df.	P
Defending Co B (177) vs Defending Co C (138)	39[b]	1.77	9	NS
Defending Co B (177) vs Attacking Co B (149)	28	0.88	26	NS
Defending Co B (177) vs Control Grp A (235)[c]	58	1.25	31	NS
Defending Co C (138) vs Control Grp A (235)[c]	97	2.15	31	< .05

[a] Measurements were taken on the Defending Co 12 hours (B) and 10 days (C) after removal from combat. The group is described in the section "Procedures and Results" in the main text.

[b] Means are based on individuals on whom paired data were obtained. This does not apply where different groups are compared. On the basis of 600 eosinophils per cu mm cutoff point, two individuals were eliminated (650 and 1250 on B; 306 and 900 on C).

[c] On the basis of 600 eosinophils per cu mm cutoff point, one individual was eliminated (890).

The British in 1952 took eosinophil counts on nine men in the British Commonwealth Division in Korea before and after going on a patrol. They observed a wide range in values (69 to 978 per cu mm before patrol) with a mean of 375 per cu mm before patrol and 310 per cu mm on return. They observed a great deal of emotional tension prior to the patrol and estimated that the patrol was not very stressful. The counts of three individuals had risen (4, 155, and 10 percent) and 6 had fallen by an average of 44 percent (13 to 73 percent) when postpatrol values were compared to prepatrol values.[16] The mean values that the British observed were higher (300+) as compared to the mean observed in this study on the Control Group (200+). In general, the results were similar showing mean poststress decreases and large individual variation.

Lymphocytes

The lymphocytes constitute about 22 percent of all white cells. They show diurnal variations with the normal range between 2000 and 3000 cells per cu mm. The level of circulating lymphocytes has been observed to decrease (lymphopenia) with stress.

Lymphopenia and eosinopenia have been observed in many of the same stress situations—exposure to cold, after adrenalin,[5(408;416)] or ACTH injection.[5(408);17]

There is a significant decline in blood lymphocytes following the glucose tolerance test. Following stressful psychomotor performance[18] (as well as after immersion for 8 minutes in 9.5° C water[19]) a fall in lymphocytes has been observed.

similarly, upon exposure to heat and high humidity, normal persons show a drop in lymphocytes.[5(411)] Lymphopenia also appears during a stress interview, with intense muscular exercise, and with acute infections.[5(411; 412; 414)]

The Control Group (Table A4) showed no significant difference between the means of lymphocyte measures taken 11 days apart, and the two measures (A and A') are significantly correlated (r= .57, significant at 1 percent level).

TABLE A4

LYMPHOCYTE COUNT, CONTROL GROUP

(per cu mm)

Mean A measure	Mean A' measure	Mean diff.	t	df.	P
2940	2849	91[a]	0.56	17	NS

[a]Means are based on 18 individuals on whom both A and A' data were available.

TABLE A5

LYMPHOCYTE COUNT, ATTACKING COMPANY

(per cu mm)

Means compared	Mean diff.	t	df.	P
Attacking Co A (3314) vs Control Grp A (2904)	410	1.70	42	.10 > .05
Attacking Co B (2539) vs Control Grp A (2904)	365	1.48	41	NS
Attacking Co C (2198) vs Control Grp A (2904)	706	3.35	42	< .01
Attacking Co D (2561) vs Control Grp A (2904)	343	1.64	39	NS
Attacking Co B (2403) vs Attacking Co D (2596)	193[a]	0.86	15	NS
Attacking Co C (2358) vs Attacking Co D (2596)	238[a]	1.19	15	NS

[a]Means are based on individuals on whom paired data were available.

TABLE A6

LYMPHOCYTE COUNT, DEFENDING COMPANY

(per cu mm)

Means compared	Mean diff.	t	df.	P
Defending Co B (2096) vs Control Grp A (2904)	808	3.39	34	< .01
Defending Co B (2096) vs Defending Co C (2485)	389[a]	1.96	11	.10 > .05
Defending Co C (2485) vs Control Grp A (2904)	419	1.77	34	.10 > .05

[a]Means are based on individuals on whom paired data were available.

Table A5 shows that the lymphocyte count in the Attacking Company, when compared to the Control Group, was not significantly lowered following stress (B measure) but did show a significant drop 5 days following combat (C). Three weeks (D) after removal from combat the lymphocyte count had increased but was not yet as high as the Control Group mean. The lymphocytes appeared to

be slower in reacting to stress than the eosinophils, and although the lymphocytes show signs of a return toward normal levels, this seemed to be quite delayed.

In the Defending Company (Table A6) the lymphocytes were clearly depressed after 5 days in combat and had returned toward normal 11 days later.

It is evident that there was a greater poststress (B) spread than on recovery. Four of the sixteen individuals in the Attacking Company showed a higher lymphocyte count following stress (B) than on recovery (D). During the same period, four others showed less than 1 standard deviation increase at recovery (D) compared to stress values (B).

Similarly, in the Defending Company, 5 of the 12 individuals showed higher stress levels (B) than recovery (C), and there was 1 whose recovery level was less than 1 standard deviation over the stress value.

Total White Count

The total white cell count shows diurnal variations, and many types of dietary deficiencies exert a pronounced effect on the white blood count. An increase in the total white cell count (leucocytosis) has been observed during

TABLE A7

TOTAL WHITE BLOOD CELL COUNT, CONTROL GROUP

(per cu mm)

Mean A measure	Mean A' measure	Mean diff.	t	df.	P
8886	9860	974[a]	1.56	17	NS

[a] Means are based on individuals on whom paired data were available.

infections and following an acute hemorrhage.[5(404; 406)] With severe stress a period of leucopenia (decrease in number) has been observed to precede the characteristic leucocytosis.[5(404-5)] (Following severe burns, leucopenia is present.)[5(407)] When white cells are markedly reduced in numbers the individual becomes quite susceptible to mouth and throat infections.

It has been known for some time that in severe stress situations there is a leucocytosis and a "shift to the left" (increase in immature neutrophils). This shift to the left has been observed when neutrophilia results from ACTH injection.[5(421)]

Table A7 shows the mean white blood cell counts for measures taken 11 days apart on the Control Groups. The means are not significantly different from each other. Therefore it is concluded that when the white cell counts of combat soldiers in a reserve area are measured at approximately the same time of day, the counts are not subject to systematic variation; however, a correlation of .22 indicates that the white blood cells as measured in a field situation are highly variable and not reliable.

The differential neutrophil count is not available on the Control Group. Instead the counts are shown (Table A8) for a group of men from an armored company at Ft Knox (March 1953) prior to going on maneuvers. This group

showed a normal Schilling Index of approximately one immature cell to five mature cells. (The Schilling Index is a ratio of immature cells—band, juvenile, and myelocyte—to mature cells, i.e., segmented.)

TABLE A8

NEUTROPHIL DIFFERENTIAL COUNT, FT KNOX

White blood cells	Mean, % of total WBC count, N = 18
Segmented cells	45.1
Band cells	9.2
Juvenile cells	-
Myelocyte cells	-
Mean Schilling Index	1/4.9

TABLE A9

TOTAL WHITE BLOOD CELL COUNT, ATTACKING COMPANY
(per cu mm)

Means compared	Mean diff.	t	df.	P
Attacking Co A (9650) vs Control Grp A (8825)	825	1.16	42	NS
Attacking Co B (6117) vs Control Grp A (8825)	2708	3.98	41	< .001
Attacking Co C (6282) vs Control Grp A (8825)	2543	4.22	42	< .001
Attacking Co D (9876) vs Control Grp A (8825)	1051	1.29	39	NS
Attacking Co B (6117) vs Attacking Co C (6277)	160[a]	0.29	18	NS
Attacking Co B (5894) vs Attacking Co D (9998)	4104[a]	6.47	15	< .001

[a] Means are based on individuals on whom paired data were available.

Contrary to expectations a leucopenia was observed in the Attacking Company (Table A9). This was due mainly to the decrease in neutrophils. Furthermore in the neutrophils there was a marked shift to the left (Table A10). This was not unexpected, but the degree of the shift, accompanied by the leucopenia is quite unusual. This type of reaction has been previously noted in terminal stages of infectious disease and in acute and severe burns.[5(404-7)] Twenty-two days after the battle (D measure) there was a reappearance of the more mature cells (Schilling Index of 1/ 1.96), although the Schilling Index had not returned to what is considered normal (see Table A8). However, the total white cell count had returned to normal.

In the Defending Company, which was composed of men in a more prolonged and sustained engagement, the same shift to the left was observed in the neutrophil differential count (Table A11), though not to the marked degree observed in the Attacking Company. No control measure on the Defending Company for the differential count was available; however, comparison can be made with the Ft Knox controls (Table A8). This shift to the left was not accompanied by a leucopenia (Table A12) at B, although there was a significant drop at C.

TABLE A 10

NEUTROPHIL DIFFERENTIAL COUNT, ATTACKING COMPANY

White blood cells	B Mean, % of total WBC count, N = 19	D Mean, % of total WBC count, N = 12
Segmented cells	13.4	39.8
Band cells	14.9	19.2
Juvenile cells	13.0	1.1
Myelocyte cells	7.5	–
Mean Schilling Index	2.64/1	1/1.96

TABLE A 11

NEUTROPHIL DIFFERENTIAL COUNT, DEFENDING COMPANY

White blood cells	B Mean, % of total WBC count, N = 13
Segmented cells	20.4
Band cells	22.4
Juvenile cells	7.5
Myelocyte cells	3.4
Mean Schilling Index	1.63/1

TABLE A 12

TOTAL WHITE BLOOD CELL COUNT, DEFENDING COMPANY

(per cu mm)

Means compared	Mean diff.	t	df.	P
Defending Co B (8304) vs Defending Co C (6875)	1429[a]	3.86	11	< .01
Control Grp A (8825) vs Defending Co B (8415)	410	0.59	35	NS
Control Grp A (8825) vs Defending Co C (6875)	1950	2.61	34	< .02

[a] Means are based on individuals on whom paired data were available.

The amount of cholesterol contained in the adrenal cortex has been shown to drop markedly under various conditions of cortical activity. The adrenals use cholesterol to manufacture hormones. "...blood constitutes an important source of cholesterol to the adrenal cortex when the gland is forced to produce large amounts of steroidal hormones for long periods of time."[20] It has been demonstrated that the cholesterol level of the blood plasma falls if the period

TABLE A13

PLASMA TOTAL CHOLESTEROL, CONTROL GROUP

(mg/100 cc)

Mean A measure	Mean A' measure	Mean diff.	t	df.	P
193.6	200	6.4[a]	1.47	17	NS

[a] Means are based on individuals on whom paired data were available.

TABLE A14

PLASMA TOTAL CHOLESTEROL, ATTACKING COMPANY

(mg/100 cc)

Means compared	Mean diff.	t	df.	P
Attacking Co A (208.9) vs Control Grp A (194)	14.9	1.48	43	NS
Attacking Co B (192.6) vs Control Grp A (194)	1.4	0.12	42	NS
Attacking Co C (193) vs Control Grp A (194)	1.0	0.09	42	NS
Attacking Co D (199.8) vs Control Grp A (194)	5.8	0.53	39	NS
Attacking Co B (192.6) vs Attacking Co C (193)	0.4[a]	-	19	NS
Attacking Co B (189.7) vs Attacking Co D (199.8)	10.1[a]	1.78	16	.10 > .05

[a] Means are based on individuals on whom paired data were available.

of cortical activity is prolonged; thus it is believed that the plasma total cholesterol levels will give an index of the magnitude of the adrenal cortical activity in stress.[19, 21] Consequently a drop in cholesterol is accompanied by an increase in 17 KS excretion.[20]

Measures of plasma total cholesterol 11 days apart on soldiers in regimental reserve (Table A13) are not significantly different and the measure is highly reliable ($r_{A-A'} = .91$ significant at the .01 level). Therefore the normal plasma cholesterol level of the combat soldier appears to be fairly stable for field studies.

The plasma total cholesterol following combat (B) in the Attacking Company (Table A14), although lower than the level 22 days later (D), was not significantly different. The mean values for plasma total cholesterol at the four measuring times, A, B, C, and D were not significantly different from the mean of the Control Group.

The values for the Defending Company in Table A15 show that following combat (B) there was a significant drop in plasma total cholesterol when compared to the Control Group or to the level 11 days later (C) in the Defending Company.

Conn et al.[20] have demonstrated that the plasma total cholesterol level decreases with increased adrenal activity as shown by an increased 17 KS excretion. The 17 KS analysis of the Korean data has been completed since the preliminary report. With this additional information it is clear that although there

TABLE A15

PLASMA TOTAL CHOLESTEROL, DEFENDING COMPANY

(mg/100 cc)

Means compared	Mean diff.	t	df.	P
Defending Co B (161.1) vs Control Grp A (194)	32.9	2.74	35	< .01
Defending Co C (194.4) vs Control Grp A (194)	0.4	0.26	34	NS
Defending Co B (164.3) vs Defending Co C (194.4)	30.1[a]	3.31	11	< .01

[a] Means are based on individuals on whom paired data were available.

was a significant decrease in plasma cholesterol in the Defending Company following combat there was no significant increase in 17 KS excretion. Thus the cholesterol decreased without an apparent increase in adrenal activity, a finding which differs from laboratory results.

Sodium/Potassium Ratio

The urine sodium/potassium (Na/K) ratio is a means of studying the effect of the adrenals on electrolyte metabolism. The expression of the sodium and potassium values as ratios eliminates the necessity of taking the large variations in absolute levels into account. Following stress there is a sodium retention and increased potassium excretion in the urine which results in lower value for the Na/K ratio.

Potassium and sodium excretion is affected by several adrenal steriods which modify kidney thresholds and change tissue electrolyte balance.[22] Increased potassium excretion is presumably occasioned by the liberation of much potassium from disintegrating cell bodies at the time of the catabolic impulse. Significant increases of potassium excretion have been noted after an hour of operation of a pursuit meter following traumatic injuries and ACTH injection.[5(196, 198)] In man, mild muscular exercise and ACTH injection cause sodium retention.[5(188)]

Sometimes the sodium/potassium picture is complicated by the lack of control of the intake of salt and water. This difficulty has been noted by Frost et al.[14] in studying the contestants in the Indianapolis Speedway race, where analysis of the data failed to show any definite pattern of response in sodium and potassium content of the urine and serum. They conclude that this could have been due to the "loss of electrolytes in the unknown volume of sweat, coupled with the impossibility of controlling the diet of the individual."

The mean difference for the Control Group for the two testing periods (Table A16) was only 0.06 and the A to A' measures were significantly correlated ($r = .44$, significant at the 5 percent level).

The mean of the urine ratios for the Attacking Company (Table A17) was significantly lower 12 hours after leaving the assault area than 5 or 22 days after leaving the assault area and also lower than the mean for the Control

TABLE A16

URINE Na/K RATIOS, CONTROL GROUP

Mean A measure	Mean A' measure	Mean diff.	t	df.	P
2.84	2.78	0.06[a]	0.30	17	NS

[a] Means are based on individuals on whom paired data were available.

TABLE A17

URINE Na/K RATIOS, ATTACKING COMPANY

Means compared	Mean diff.	t	df.	P
Attacking Co B (1.9) vs Attacking Co C (3.4)	1.50	2.97	16	< .01
Attacking Co B (1.9) vs Attacking Co D (3.7)	1.80	3.61	16	< .01
Attacking Co C (3.4) vs Attacking Co D (3.7)	0.30	0.80	16	NS
Control Grp A (3.07) vs Attacking Co B (2.01)	1.06	3.42	42	< .01
Control Grp A (3.07) vs Attacking Co C (3.51)	0.44	1.41	42	NS
Control Grp A (3.07) vs Attacking Co D (3.72)	0.65	1.71	39	.10 > .05
Control Grp A (3.07) vs Attacking Co A (2.04)	1.03	4.42	46	< .001

Group. Such findings indicate adrenal cortical activity with respect to electrolytes and are in accord with the present views of stress physiology. The Na/K ratios had returned to normal some 5 days after leaving the assault area (C).

The Defending Company showed a different reaction (Table A18). Following removal from combat this group had a high mean ratio indicating hypoadrenal activity. This was not significantly higher than the mean ratio 11 days later but was significantly higher than the Control Group mean. Also the mean ratio of the Defending Company was significantly higher than that of the Attacking Company.

The plasma Na/K ratios are not presented because a breakdown of the blood cells which affected the potassium values was encountered in preservation of the samples. Saliva values were unusable because of the highly unreliable means of collecting saliva even in a controlled situation.

Urinary Chloride

Total chloride, like sodium and potassium is a constituent of the urine which reflects the effect of the adrenal hormones on electrolyte metabolism. Chloride excretion in the urine is a means of determining the degree to which

salt-retaining steroids have been activated. A decrease in urinary elimination has been observed with patients suffering from extensive burns and after ACTH injection.[5(136; 191)] In general the changes in chloride excretion follow those observed in sodium, i.e., are confirmatory to sodium and potassium. However, when liquid intake is not controlled the Na/K ratio is a better measure of

TABLE A 18

URINE Na/K RATIOS, DEFENDING COMPANY

Means compared	Mean diff.	t	df.	P
Defending Co B (4.9) vs Defending Co C (3.4)	1.50	1.72	11	NS
Control Grp A (3.07) vs Defending Co B (5.15)	2.08	3.98	35	< .001
Control Grp A (3.07) vs Defending Co C (3.37)	0.30	0.71	34	NS
Defending Co B (5.15) vs Attacking Co B (2.01)	3.14	4.90	31	< .001

TABLE A 19

URINARY CHLORIDE, CONTROL GROUP

(meq/hr)

Means compared	Mean diff.	t	df.	P
Control Grp A (13.6) vs Control Grp A' (10.7)	2.9[a]	2.02	17	NS

[a] Means are based on individuals on whom paired data were available.

TABLE A 20

URINARY CHLORIDE, ATTACKING COMPANY

(meq/hr)

Means compared	Mean diff.	t	df.	P
Attacking Co A (11.4) vs Control Grp A (13.5)	2.1	1.29	42	NS
Attacking Co B (5.6) vs Control Grp A (13.5)	7.9	5.61	42	< .001
Attacking Co C (10.9) vs Control Grp A (13.5)	2.6	1.46	42	NS
Attacking Co D (13.0) vs Control Grp A (13.5)	.5	—	39	NS
Attacking Co B (5.6) vs Attacking Co C (10.9)	5.3[a]	2.68	19	< .02
Attacking Co B (5.8) vs Attacking Co D (13.0)	7.2[a]	3.43	16	< .01

[a] Means are based on individuals on whom paired data were available.

adrenal activity since chloride would increase with increased excretion but sodium and potassium being expressed as a ratio would not be affected by volume changes.

Table A19 shows that total chloride excretion indicated in measures taken 11 days apart on soldiers in the reserve area is not significantly different. The A to A' measures are significantly correlated ($r = .47$, significant at the 5 percent level). Therefore chloride excretion is a reliable measure and is not altered systematically in a control situation.

From Table A20 it is clear that following stress (B) the Attacking Company showed a significant reduction in chloride excretion when compared with the mean 5 days (C) and 22 days (D) following combat, and with the Control Group.

As shown in Table A21 the Defending Company shows a significantly low mean value for chloride excretion following combat when compared with the Control Group mean. The mean at B is not significantly lower than the mean 11 days following combat (C). The mean at C for both the Attacking and Defending Companies (11 meq/hr) is lower than the Control Group and the Attacking Company D measures (13 meq/hr) suggesting that chloride excretion had not returned to normal.

In general the Na/K ratios are correlated with chloride excretion under stress as shown in Table A22.

TABLE A21

URINARY CHLORIDE, DEFENDING COMPANY

(meq/hr)

Means compared	Mean diff.	t	df.	P
Defending Co B (8.0) vs Control Grp A (13.5)	5.5	2.89	33	.01
Defending Co C (10.5) vs Control Grp A (13.5)	3.0	1.72	33	.10 > .05
Defending Co B (8.0) vs Defending Co C (10.5)	2.5[a]	0.98	10	NS

[a] Means are based on individuals on whom paired data were available.

TABLE A22

CORRELATION BETWEEN CHLORIDE EXCRETION AND Na/K RATIOS

(meq/hr)

Comparison	rho	PE
Defending Co B	.52	±.149
Attacking Co C	.49	±.120
Control Grp A	.20	±.087

Nitrogen

Blood urea, urinary urea, uric acid, and creatinine are nitrogenous end products of protein metabolism in the body. Creatine is important in muscle contraction and recovery of contractibility. It is eliminated in the urine as creatinine. Normally the amount excreted is quite constant. Increases in creatinine excretion have been observed during the early stages of stress. Such stressors as electro-shock, burns, and fractures can cause marked creatinine excretion, presumably as a result of sudden tissue catabolism.[5(177)]

Uric acid reflects the conversion of protein to sugar by action of the steroids. It is suspected that the pronounced decomposition of lymphocytes which occurs with stress represents an important source of the liberated uric acid. With injection of ACTH there is a sharp increase in urinary uric acid[5(181, 265, 269)] which may be explained by a combination of increased uric acid production and lowered renal threshold.

A negative nitrogen balance (increased nitrogen excretion) follows traumatic injury, surgery,[23] and ACTH administration; cold, muscular exhaustion,[5(150)] fractures, burns, and infections.[8] Subjects performing on a pursuit meter have shown significant increases in creatinine and urinary uric acid output. Similar results have been recorded for a target-ball test.[24]

The mean values in the Control Group (Table A23) showed a significant drop on second testing in all nitrogen measures.* In spite of this the Control Group may still be used for comparison with the stress groups. Since with stress there is an expected increased nitrogen excretion, the stress measures were compared with the highest control value (A). On this basis any significant increase with stress would be real and would not be a function of a depressed mean control value.

The mean values for the three nitrogen products in the Attacking Company (Table A23) following combat (B) were significantly higher than their own control values (C) and the highest Control Group value (A). It should be pointed out that in B of the Attacking Company the increased urea nitrogen excretion was reflected in the blood with a very significantly high blood urea value; almost a 20-mg percent urea nitrogen in the blood. Therefore it is evident that there was protein catabolism. The Defending Company, on the other hand, showed no shift on stress data (B) from either their own control measure (C) or the Control Group, with the exception of creatine. In the latter case there was a significant mean increase in creatinine 11 days after leaving the assault area, although this value (C) does not differ significantly from the Control Group mean.

It would take approximately 2 days without food to produce protein catabolism such as that observed in the Attacking Company. These men were not without food during their 18 hours in combat, and therefore diet did not account fully for the nitrogen excretion observed. In addition, if the nitrogen excretion were due to diet the 17 KS excretion would be one-third the value observed in the Attacking Company. The Attacking Company therefore showed a significant protein catabolism due to combat stress whereas the Defending Company had normal protein metabolism.

A test of the hypothesis of homogeneity of variance was applied because it appeared that the variance of the Attacking Company was much larger than either the Defending Company or the Control Group. From Table A24 it is apparent that the Attacking Company did have a significantly larger variance than either of the other two groups on all three of the measures of nitrogen metabolism. The effect of stress on the Attacking Company caused an increase in the variability of the group on the measures of nitrogen metabolism.

There was a consistent high correlation in the Attacking Company (B) on the three measures of nitrogen metabolism (Table A25). Any one of the three would be sufficient to indicate nitrogen catabolism. On the other hand, creatinine in

*Urea, uric acid, and creatinine are reliable measures. They were significantly correlated from A to A' (urea .54; uric acid .41; creatinine .73; all significant at the 5 percent level).

TABLE A 23

COMPARISON OF GROUP MEANS ON URINARY UREA, URIC ACID, AND CREATININE (mg/hr)

Measure	Means compared		Mean diff.	t	df.	P
	Control Grp A vs Control Grp A[a]					
Urea	498.2	420.0	78.2	2.37	17	< .05
Uric acid	38.2	27.6	10.6	5.31	17	< .001
Creatinine	77.8	66.6	11.2	3.64	17	< .01
	Attacking Co B vs Attacking Co C[a]					
Urea	878.4	423.0	455.4	3.42	19	< .01
Uric acid	53.6	35.1	18.5	2.51	19	< .05
Creatinine	121.4	67.3	54.1	2.63	19	< .02
	Attacking Co B vs Attacking Co D[a]					
Urea	884.1	552.8	331.3	2.15	16	.05
Uric acid	56.4	36.9	19.5	2.05	16	.10 > .05
Creatinine	124.6	77.4	47.2	1.98	16	.10 > .05
	Attacking Co C vs Attacking Co D[a]					
Urea	442.9	552.8	109.9	2.22	16	< .05
Uric acid	37.2	36.9	0.35	0.10	16	NS
Creatinine	70.3	77.4	7.1	0.35	16	NS
	Defending Co B vs Defending Co C[a]					
Urea	486.9	581.0	94.1	1.24	10	NS
Uric acid	33.1	36.4	3.3	0.87	10	NS
Creatinine	69.8	85.1	15.3	2.89	10	< .02
	Control Grp A vs Attacking Co B					
Urea	495.2	878.4	383.2	3.05	42	< .01
Uric acid	37.8	53.6	15.8	1.99	42	.10 > .05
Creatinine	80.2	121.4	41.2	2.40	42	< .05
	Control Grp A vs Defending Co B					
Urea	495.2	486.9	8.3	0.15	33	NS
Uric acid	37.8	33.1	4.7	0.90	33	NS
Creatinine	80.2	69.8	10.4	1.63	33	NS
	Control Grp A vs Attacking Co B					
Urea	495.2	672.4	177.2	2.66	42	< .02
Uric acid	37.8	47.5	9.7	2.31	42	< .05
Creatinine	80.2	103.5	23.3	2.62	42	< .02
	Defending Co B vs Attacking Co B					
Urea	486.9	876.1	389.2	2.16	29	< .05
Uric acid	34.1	53.6	19.5	1.65	29	NS
Creatinine	69.8	121.4	51.6	1.82	29	.10 > .05

[a] Means are based on individuals on whom paired data were obtained.

the Deferding Company presented a somewhat different picture than either urea or uric acid, and the correlations with creatinine were low.

Urinary Steroids and Corticoids

Accurate estimates of the function of the adrenal cortex may be made by measuring the amount of actual adrenal hormones and the end products of adrenal hormone metabolism which appear in the urine. This study involved measurement of the Porter-Silber (P.S.) chromogens (which include among other things,

TABLE A 24

F RATIOS OF GROUPS FOR URINE UREA, URIC ACID, AND CREATININE (mg/hr)

Measure	Variance compared		df.	F	P
	Attacking Co B vs Defending Co B				
Urea	342,909	47,006	19 and 10	7.30	< .02
Uric acid	1,396	206	19 and 10	6.78	< .02
Creatinine	8,537	291	19 and 10	29.3	< .002
	Attacking Co B vs Control Grp A				
Urea	342,909	27,909	19 and 23	12.29	< .002
Uric acid	1,396	92	19 and 23	15.20	< .002
Creatinine	8,537	314	19 and 23	27.20	< .002
	Defending Co B vs Control Grp A				
Urea	47,006	27,909	10 and 23	1.68	NS
Uric acid	206	92	10 and 23	2.24	NS
Creatinine	291	314	10 and 23	1.08	NS

TABLE A 25

CORRELATION OF STRESS MEASURES (B) OF UREA, URIC ACID, AND CREATININE IN ATTACKING AND DEFENDING COMPANIES

Measure	Attacking Co	Defending Co
	rho	rho
Urea vs uric acid	.89	.91
Urea vs creatinine	.34	.44
Uric acid vs creatinine	.83	.23

the actual hormones), and 17 ketosteroids (KS) (the metabolic end products of adrenal and some other hormones).

Following exposure to stress, 17 KS elimination usually increases. If the stress is continued, the 17 KS output falls below normal values.[25,26] In a study of 66 patients who suffered medical or surgical injury it was found that "...urinary 17 KS excretion usually rises for a brief period (1 to 3 days) and almost always falls...until convalescence is achieved, when it gradually returns to normal."[27]

Many other stressers have been found to give rise to similiar 17 KS changes, e.g., flight testing,[28,29] speedway racing,[14] a submarine-tank stress situation,[30] emotional stress,[29] irradiation, electro-shock, and some drugs.[5(225-27)]

The P.S. chromogens composed of the adrenal hormones and some metabolic by-products are expected to follow a similar pattern to that of the 17 KS. It is thought by some that since the P.S. chromogens contain some of the actual hormone they provide a better estimate of adrenal activity than the 17 KS. This is questionable, however, since there are greater problems involved in making the actual determinations. The P.S. chromogens and 17 KS should essentially substantiate one another, and together provide a better estimate of what is happening to the adrenal gland than either one would alone.

TABLE A 26

17 KS, ATTACKING COMPANY

(mg/hr)

Means compared	Mean diff.	t	df.	P
Control Grp A (0.64) vs Attacking Co B (0.93)	0.29	1.67	42	NS
Control Grp A (0.64) vs Attacking Co A (0.69)	0.05	0.60	42	NS
Attacking Co A (0.69) vs Attacking Co B (1.01)	0.32	1.45[a]	34	NS
Attacking Co B (0.96) vs Attacking Co C (0.61)	0.35	2.03[b]	18	.06
Attacking Co B (0.96) vs Attacking Co D (0.72)	0.24	0.09[b]	12	NS

[a] Individuals on whom paired data were available were not included in this test.
[b] Only individuals on whom paired data were available were used.

The method for extraction of 17 KS from urine was that described by Pincus.[31] The urine was hydrolyzed and extracted with ether and prepared to the point of Girard separation in Korea. The extracts in test tubes were flown to the United States, and the Girard separation was accomplished at the laboratories of the Worcester Foundation for Experimental Biology. The data to be presented here include only the ketonic fraction of neutral steroids. Samples represent urine collections over a mean time of 3 hours during approximately the same time of day.

Table A26 presents the mean 17 KS output for the groups studied and shows the statistical significance of the differences between compared pairs of these means. The Control Group measures of 0.64 mg/hr compare favorably with the many measures taken under normal conditions in this country with the normal range considered to be between 0.50 and 0.70 mg/hr. The values of the stress groups several days following combat [Attacking Company (C) and Defending Company (C)] were also within the normal range, being 0.61 and 0.50 mg/hr, respectively. The Attacking Company (B) (the poststress sample) was significantly higher than its later value (C) (at the .06 confidence level) indicating an increase in 17 KS output as a result of stress.

In contrast the Defending Company (B) (the poststress sample) (Table A27) was significantly lower than the Control Group and also significantly lower than the Attacking Company (B). This, contrary to the Attacking Company, indicates a lowering of 17 KS following stress.

An examination of the variances of the measures of the groups further emphasizes the differences just noted.

It can be seen from Table A28 that the F test shows the variance of the Attacking (B) to be significantly higher than all control measures and those of the Defending Company (B). This significantly higher variance in B of the Attacking Company indicates that combat stress in some fashion operated differentially on the individuals insofar as their 17 KS output was concerned. The extremely high variance at B of the Attacking Company may account for the fact that even though the mean poststress 17 KS output for the Attacking Company was higher than all other measures, it was not significantly higher in all instances.

TABLE A27

17 KS, DEFENDING COMPANY
(mg/hr)

Means compared	Mean diff.	t	df.	P
Control Grp A (0.64) vs Defending Co B (0.39)	0.25	3.40	34	< .01
Attacking Co B (0.93) vs Defending Co B (0.39)	0.54	2.81[a]	30	< .01
Defending Co B (0.42) vs Defending Co C (0.50)	0.08	1.32[b]	10	NS

[a] Corrected for significant "F" by method of Cochran and Cox (W.G. Cochran and G. Cox, "Experimental Design," mimeographed, 1944).
[b] Only individuals on whom paired data were available were used.

TABLE A28

VARIANCE OF 17 KS
(mg/hr)

Variances compared	df.	F	P
Control Grp A (0.0416) vs Attacking Co A (0.1206)	23 and 19	2.90	.02
Control Grp A (0.0416) vs Attacking Co B (0.6919)	23 and 19	16.63	.002
Control Grp A (0.0416) vs Attacking Co C (0.1238)	23 and 18	2.98	.02
Control Grp A (0.0416) vs Attacking Co D (0.0606)	23 and 12	1.46	NS
Control Grp A (0.0416) vs Defending Co B (0.0363)	23 and 11	1.15	NS
Control Grp A (0.0416) vs Defending Co C (0.0105)	23 and 11	3.96	.02
Attacking Co A (0.1206) vs Attacking Co B (0.7875)	19 and 16	6.53	.002
Attacking Co B (0.6919) vs Attacking Co C (0.1238)	19 and 18	5.59	.002
Attacking Co B (0.6919) vs Defending Co B (0.0363)	19 and 11	19.06	.002

The P.S. chromogens were extracted from the same urine samples which provided the 17 KS determination, by a modified Porter-Silber method.[32] The chromogens were extracted with butanol and evaporated to dryness in Korea. The dried samples were then flown to the United States, and the colorimetry was performed by Dr. Peter Forsham at the University of California Metabolic Institute.

Table A29 presents the mean P.S. chromogen data for the groups studied. Owing to the small number of determinations accomplished in each group, no statistical interpretation was attempted.

It can be seen from the data that these means generally support the data obtained on 17 KS above. The mean of the Attacking Company at B was only slightly higher than the recovery measures, whereas the mean of the Defending Company at B was the same as or lower than the control values. However, again, it should be noted that there is a large variation in the Attacking Company at B but a considerably smaller variance in the Defending Company at B (see annex to this appendix).

TABLE A29

MEANS OF PORTER-SILBER CHROMOGENS

(mg/hr)

Group	Times of measurement				
	A	A'	B	C	D
Control Grp	0.27 (2)[a]	0.36 (3)[a]	-	-	-
	(A + A') mean = 0.32 (5)[a]				
Attacking Co	0.33 (6)[a]	-	0.72 (7)[a]	0.50 (12)[a]	0.49 (7)[a]
Defending Co	-	-	0.31 (7)[a]	0.69 (6)[a]	-

[a] N is shown in parentheses.

ACTH Test

It has been established that adrenocorticotrophic hormone (ACTH), a protein hormone from the anterior putuitary gland, activates the adrenal cortex, which in turn releases steroid hormones, principally Compound F. A measure of the adrenal cortical function during stress can be made by measuring the amount of actual hormones secreted and the metabolic end products of these hormones excreted in the urine (17 KS, P.S. chromogens) and also by measuring the effect of the hormones on the tissues and organs (Na/K ratio, chloride, urea, uric acid, and creatinine). In addition, ACTH can be injected to determine from the hormones, metabolic end products, and tissue breakdown just what the reserve of the adrenals is following stress. If the adrenal glands are depleted of hormones following stress, then injection of ACTH should result in less corticoid excretion and less secondary effect on the electrolytes and nitrogen metabolism than the same test on the same subject following recovery. It is important to determine the reserve capacity of the adrenal glands, for the organism is able to withstand added insult if the gland is functioning properly; if not, such ability is lost. Therefore the ACTH test is used to evaluate the adrenal cortical reserve by stimulating the gland to its utmost capacity and measuring the response.

The method of administering the ACTH test involved giving a standard dose of 2.0 ml of ACTH in gel administered intramuscularly into the gluteus maximus immediately following the test battery. The subject voided just before the injection and the time was recorded. He was told to collect all his urine from then on in a liter bottle containing 5 ml of 3 percent thymol in glacial acetic acid. The period of collection extended from early evening until 0630 the following morning. The subject himself recorded on the bottle the last time of voiding. The total period of collection was approximately 12 hours.

The effect on the electrolytes, nitrogen metabolism, and hormone excretion has been analyzed to evaluate the responsiveness of the adrenal gland following combat. The results were not treated statistically since most samples include six or fewer subjects. The means are presented in graphic form with individual values tabled.

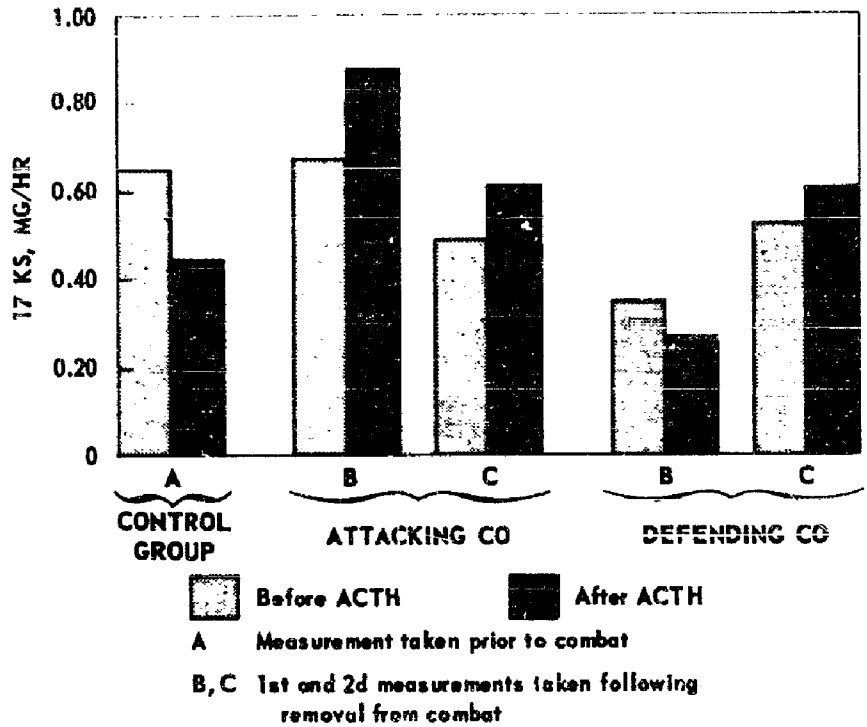


Fig. A1—Mean 17 KS before and after ACTH Administration

17 Ketosteroids. Figure A1 and Table A30 compare the mean 17 KS output before and after ACTH administration for the various groups under the different test conditions.

It was expected that the injection of ACTH in normal subjects would stimulate the secretion of adrenal hormones which in turn would give rise to an increased excretion of 17 KS. In the control subjects this was not the case; there was not an increase in 17 KS output following ACTH administration. This may be accounted for by the fact that the 17 KS before ACTH were determined from samples collected during the day, whereas the post-ACTH 17 KS represented overnight collections. The apparent lack of stimulation, then, is most likely due to the failure of the ACTH to overcome completely the known decline in 17 KS output during sleep.[33]

Figure A1 and Table A30 also show the increase in 17 KS excretion in response to ACTH by the Attacking Company immediately following combat (B). This output of 17 KS is greater than that caused by combat stress as indicated by the pre-ACTH measure. In contrast to the post-ACTH normal values in the Attacking Company are the low values in post-ACTH 17 KS in the Defending Company, confirming the finding of low adrenal activity in this group following combat. It should be noted that both the Attacking and Defending Companies responded to ACTH with an increased 17 KS excretion when they were measured

5 to 11 days after combat, at which time it can be assumed they were approaching recovery; however, the adrenal may still be somewhat hypersensitive, accounting for the difference in reaction of these groups from that of the Control Group.

In both the Attacking and Defending Companies immediately following combat the high 17 KS excreters before ACTH are high following ACTH, whereas the low excreters before ACTH injection fail to be stimulated by the ACTH.

TABLE A30

17 KS BEFORE AND AFTER ACTH ADMINISTRATION
(mg/hr)

Subjects, Control Grp	A	
	Before	After
H9329	0.55	0.47
H8421	0.42	0.46
H3136	0.72	0.68
M7371	0.59	0.74
W4130	0.63	0.28
W7018	0.89	0.41
S1495	0.65	0.23
W1009	0.64	0.28
Mean	0.64	0.44

Subjects, Attacking Co	B		C	
	Before	After	Before	After
H4808	1.01	1.75	0.93	0.79
M0305	0.32	0.39	0.13	0.51
S4171	0.45	0.59	0.48	0.75
W1083	0.88	0.73	0.42	0.42
Mean	0.67	0.87	0.49	0.62

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	-	0.42	0.63	0.77
R0371	0.45	0.26	0.46	0.48
S4749	0.52	0.33	0.40	0.51
T1482	0.32	0.16	0.59	0.59
W7805	0.07	0.16	0.59	0.61
Mean	0.34	0.27	0.53	0.61

P.S. Chromogens. Figures A2 and A3, supported by Tables A31 and A32, respectively, show the results obtained by measuring P.S. chromogens in the urine before and after ACTH administration under the previously described testing conditions. Figure A2 and Table A31 show all the Porter-Silber determinations, and Fig. A3 and Table A32 describe the data from subjects from whom repeat data were obtained.

The Porter-Silber data generally support the data obtained by measurement of the 17 KS. There was no response to ACTH in the Defending Company at B, again indicating no adrenal activity. The response of the Attacking Company following combat was lower after ACTH than before ACTH, as shown in

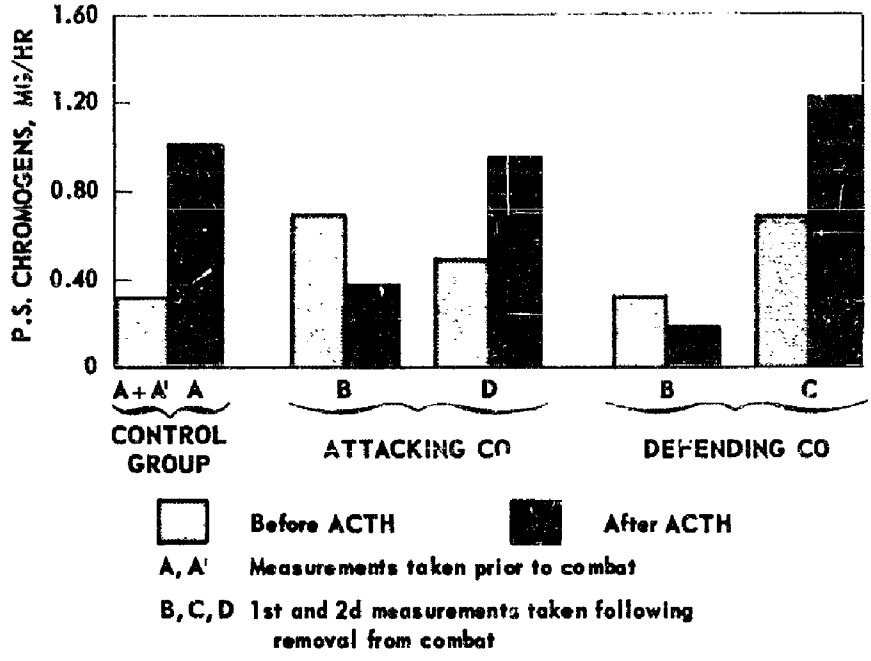


Fig. A2—Mean P.S. Chromogens Based on Complete Determinations before and after ACTH Administration

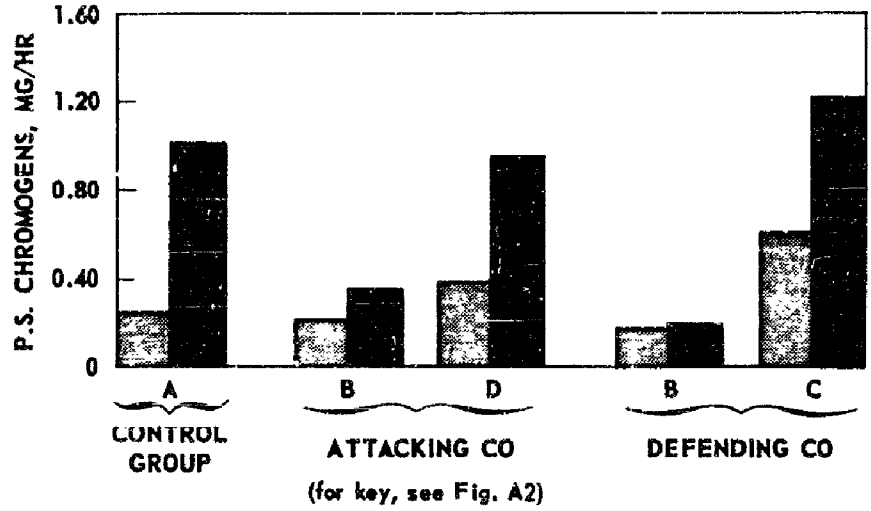


Fig. A3—Mean P.S. Chromogens on Subjects for Whom Repeat Data Were Partially Complete before and after ACTH Administration

Fig. A2, although the value was somewhat higher than obtained in the Defending Company. Unfortunately there were small numbers of post-ACTH determinations in both groups, because only a portion of the total samples were analyzable. It should be pointed out that of the three individuals in the Attacking Company

TABLE A31

COMPLETE P.S. CHROMOGENS
BEFORE AND AFTER ACTH ADMINISTRATION
(mg/hr)

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H8421	–	1.5	–	–
H3136	–	0.92	0.32	–
W7018	0.28	0.64	–	–
W1495	0.25	–	–	–
M7371	–	–	0.27	–
W4130	–	–	0.48	–
Mean	0.27	1.02	0.36	–

Mean combined data, A + A' before ACTH = 0.32

Subjects, Attacking Co	B		D	
	Before	After	Before	After
C4136	0.32	–	–	–
H4808	0.34	0.37	0.26	1.40
M0305	0.13	0.25	0.45	0.78
P4185	0.96	–	0.34	–
S4171	–	0.50	–	0.90
T9985	0.81	–	–	–
W2990	2.30	–	–	–
W1083	0.19	–	0.47	0.74
H7321	–	–	0.84	–
H1864	–	–	0.72	–
H1296	–	–	0.37	–
Mean	0.72	0.37	0.49	0.96

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	–	–	1.1	–
B5392	0.30	–	–	–
J4476	0.24	–	–	–
L0624	0.82	–	1.0	–
P0411	0.29	–	–	–
R0371	–	–	0.92	1.12
S4749	0.45	0.39	0.29	1.20
T1483	0.08	0.024	0.29	1.71
W7805	0.02	0.16	0.56	0.83
Mean	0.31	0.19	0.69	1.22

from whom post-ACTH data were obtained following combat, the two for which pre-ACTH values were available were low P.S. chromogen excreters prior to ACTH. Since the 17 KS data indicate that those who were low before ACTH also gave a low response to ACTH, the fact that the Attacking Company's post-ACTH

TABLE A 32

P.S. CHROMOGENS BEFORE AND AFTER ACTH ADMINISTRATION ON SUBJECTS FOR WHOM REPEAT DATA WERE PARTIALLY COMPLETE

(mg/hr)

Subjects, Control Grp	A	
	Before	After
H8421	-	1.5
H3136	-	0.92
W7018	0.28	0.64
S1495	0.25	-
Mean	0.27	1.02

Subjects, Attacking Co	B		D	
	Before	After	Before	After
H4808	0.34	0.37	0.26	1.40
M0305	0.13	0.25	0.45	0.78
S4171	-	0.50	-	0.90
W1083	0.19	-	0.47	0.74
Mean	0.22	0.37	0.39	0.96

Subjects, Defending Co	B		C	
	Before	After	Before	After
R0371	-	-	0.92	1.12
S4749	0.45	0.39	0.29	1.20
T1483	0.08	0.024	0.29	1.71
W7805	0.02	0.16	0.56	0.83
Mean	0.18	0.19	0.52	1.22

data were obtained on only three individuals, two of whom were low excreters prior to ACTH, undoubtedly accounts for the low P.S. chromogen response to ACTH by this company.

The data of the Control Group and of the Attacking and Defending Companies 11 to 22 days after combat followed the usual pattern. There was in the Defending and Attacking Companies a dramatic increase in response to ACTH in contrast to the marked low response at B.

Electrolytes. Figure A4 shows graphically the Na/K ratio before and after ACTH and Table A33 shows the actual numerical ratios. Figure A5 and Table A34 present the urinary total chloride data. In the Control Group both the Na/K ratio and total chloride dropped approximately the same amount on A and A', indicating adrenal sensitivity. In B of the Attacking Company the adrenal responded to ACTH even though the pre-ACTH samples already indicated increased adrenal activity. In C sample, the Na/K ratio and urinary chloride values were normal in pre-ACTH samples and reduced in post-ACTH samples as expected.

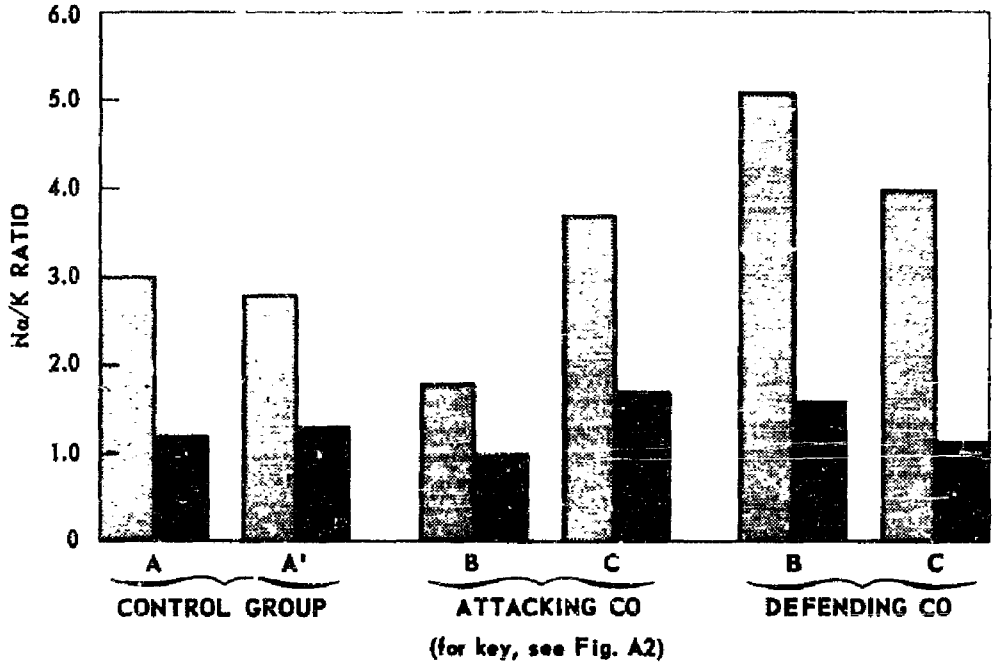


Fig. A4—Mean Urinary Na/K Ratios before and after ACTH Administration

In contrast the Defending Company at B had a high Na/K ratio indicating hypoadrenal activity. On administration of ACTH, however, there was a marked lowering of the Na/K ratio and urinary chloride indicating some activity of the adrenal gland with respect to electrolytes. The pre- and post-ACTH responses at C were comparable to the Attacking Company and the Control Group.

Nitrogen. A negative nitrogen balance (increased nitrogen excretion) is expected following successive injection of ACTH over a period of days. Ingle concludes from his work with adrenalectomized or sham-operated rats that adrenocortical hormones are a required, but not causal, factor for increased nitrogen excretion.[9]

The pre- and post-ACTH data for urea, uric acid, and creatinine for the three groups at the various measurement times are shown in Figs. A6 to A8 and Tables A35 to A37. The Control Group showed a mean decrease on the nitrogen measures following ACTH administration. The individual values showed no consistency—some increased, others decreased following ACTH.

In the Attacking Company the mean urea, uric acid, and creatinine excretions at B following ACTH were considerably lower than the pre-ACTH values at B. Each individual showed this drop. At C and D the post-ACTH means were

slightly lower although there was no consistency among individuals. The Defending Company, on the other hand, showed a small post-ACTH mean increase at B although there were some individuals who showed decreases.

TABLE A33

URINARY Na/K RATIOS
BEFORE AND AFTER ACTH ADMINISTRATION

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H9329	3.6	1.3	-	-
H8421	2.9	0.8	3.1	0.9
H3136	3.8	0.8	4.3	1.6
M7371	2.8	1.9	2.1	1.5
W4130	2.5	1.5	2.7	1.3
W7018	3.8	0.8	-	-
S1495	2.7	1.0	2.9	1.6
W1009	1.8	1.4	1.7	1.0
Mean	3.0	1.2	2.8	1.3

Subjects, Attacking Co	B		C	
	Before	After	Before	After
S4171	2.7	0.7	2.8	1.8
W1083	1.0	1.4	4.6	1.9
H4808	1.4	0.4	3.9	1.1
M0305	2.1	1.5	3.6	1.9
Mean	1.8	1.0	3.7	1.7

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	3.1	1.7	8.4	2.4
R0371	3.3	1.3	2.5	0.7
S4749	3.8	1.4	3.5	1.4
T1483	8.2	2.0	3.2	0.6
W7805	6.9	1.7	2.5	0.9
Mean	5.1	1.6	4.0	1.2

In general the results with ACTH failed to show a consistent change. The results may be partly explained on the basis that the ACTH urine was collected overnight at a time when nitrogen output would normally be low; the ACTH stimulation was sufficient to restore the decline in nitrogen excretion occuring during sleep but not sufficiently prolonged to obtain the catabolic response. However, the increase in nitrogen excretion in response to ACTH at B in the Defending Company cannot be explained adequately at the present time.

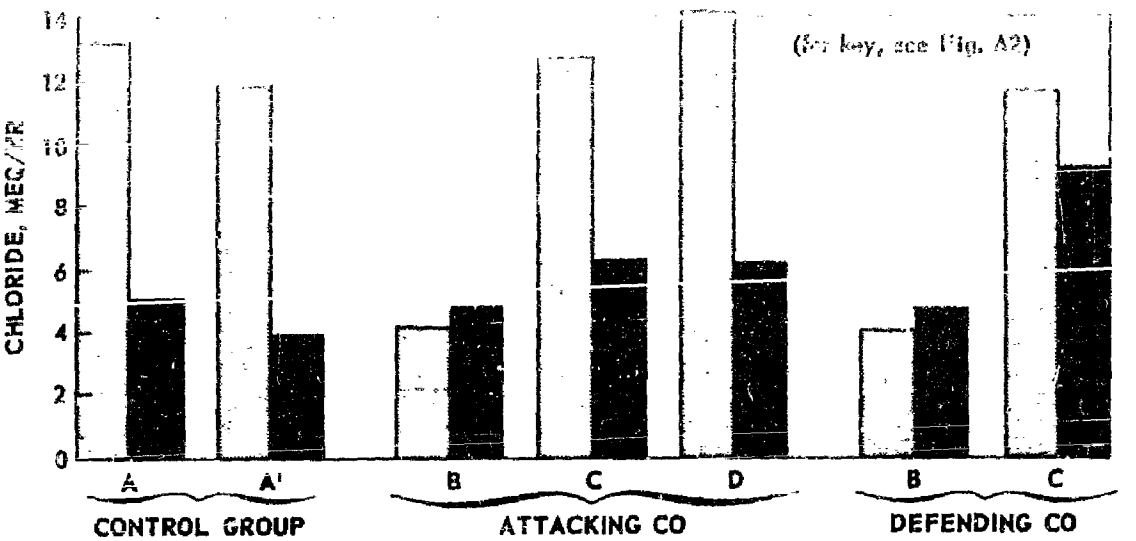


Fig. A5—Mean Urinary Chloride before and after ACTH Administration

TABLE A34

URINARY CHLORIDE BEFORE AND AFTER ACTH ADMINISTRATION

(meq/hr)

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H9329	16.7	4.8	-	-
H8421	13.4	5.5	8.1	3.5
H3136	15.4	3.1	17.8	1.6
M7371	8.8	10.0	8.1	4.6
W4130	15.2	5.5	11.5	3.5
W7018	18.5	2.6	-	-
S1495	5.2	3.0	11.2	5.2
W1009	7.9	5.6	14.6	4.2
Mean	12.6	5.0	11.9	3.8

Subjects, Attacking Co	B		C		D	
	Before	After	Before	After	Before	After
H4808	2.3	7.9	14.4	7.7	17.4	6.8
M0305	4.0	2.9	3.9	6.3	6.0	4.1
S4171	6.7	5.1	6.1	5.4	11.8	8.9
W1083	4.0	3.4	25.9	6.0	21.0	5.1
Mean	4.3	4.8	12.6	6.4	14.1	6.2

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	1.2	3.3	22.1	13.1
R0371	4.8	4.1	7.5	6.4
S4749	3.7	7.2	8.9	13.8
T1483	7.7	4.3	10.5	7.2
W7805	2.7	4.3	9.1	5.7
Mean	4.0	4.6	11.6	9.2

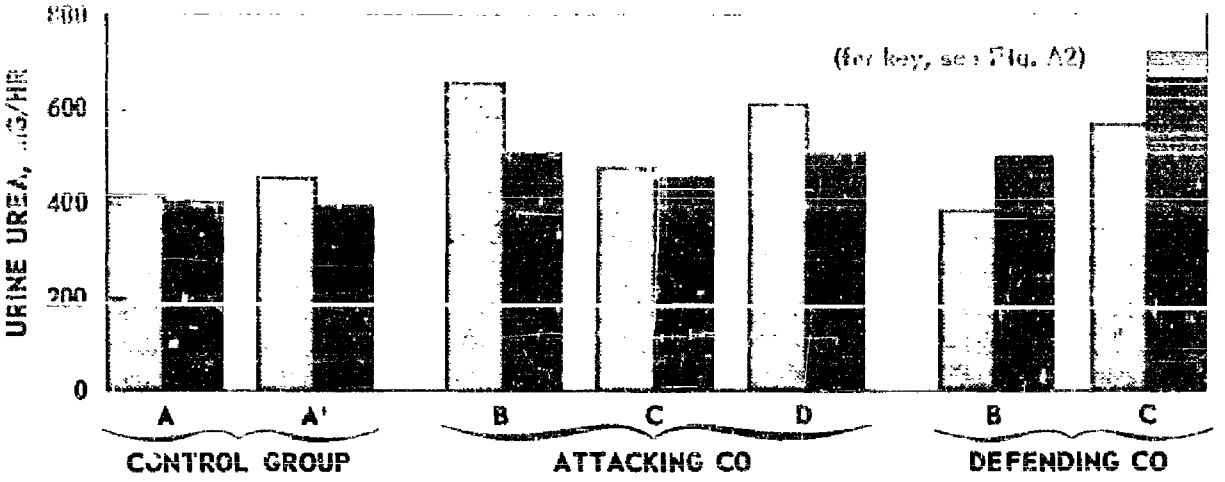


Fig. A6—Mean Urine Urea before and after ACTH Administration

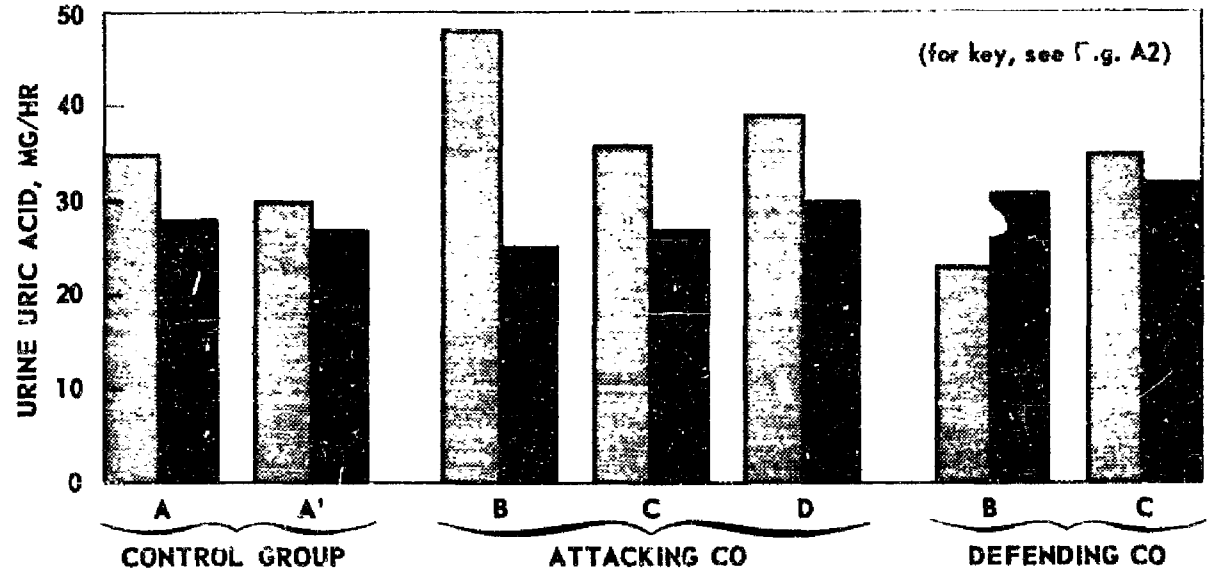


Fig. A7—Mean Urine Uric Acid before and after ACTH Administration

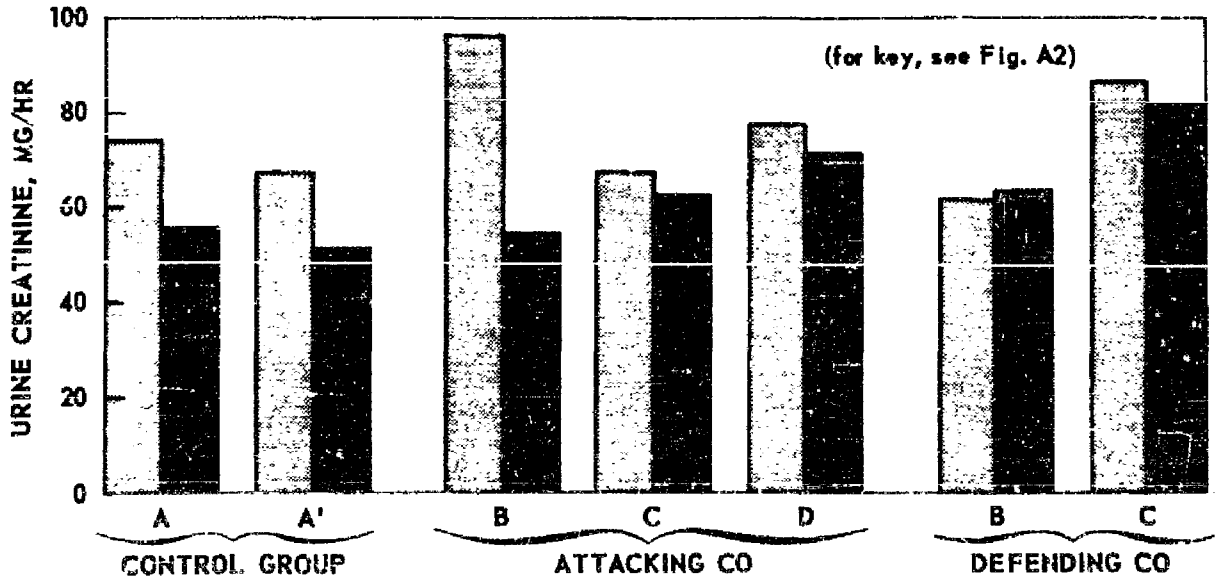


Fig. A8—Mean Urine Creatinine before and after ACTH Administration

TABLE A 35

URINARY UREA BEFORE AND AFTER ACTH ADMINISTRATION

(mg/hr)

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H9329	537	428	-	-
H8421	507	533	566	517
H3136	646	481	657	541
M7371	386	604	331	297
W4130	483	404	380	463
W7018	241	420	-	-
S 1495	371	286	335	354
W1009	294	202	521	210
Mean	433	420	465	399

Subjects, Attacking Co	B		C		D	
	Before	After	Before	After	Before	After
H4808	571	527	260	476	654	723
M0305	444	366	308	333	404	471
S 4171	543	471	557	590	688	508
W1083	1077	740	771	434	698	356
Mean	659	526	474	458	611	515

Subjects, Defending Co	B		C	
	Before	After	Before	After
F9576	198	522	742	799
R0371	405	421	500	572
S 4749	461	672	411	851
T1483	454	378	589	627
Mean	380	498	561	712

TABLE A 36

URINARY URIC ACID BEFORE AND AFTER ACTH ADMINISTRATION

(mg/hr)

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H9329	40	26	-	-
H8421	33	43	34	35
H3136	33	30	36	26
M7371	23	40	23	22
W4130	40	27	31	37
W7018	44	20	-	-
S 1495	37	19	23	31
W1009	28	17	32	12
Mean	35	28	30	27

Subjects, Attacking Co	B		C		D	
	Before	After	Before	After	Before	After
H4808	51	20	38	38	38	50
M0305	25	8	15	14	23	13
S4171	50	38	45	30	43	36
W1083	67	32	46	24	51	20
Mean	48	25	36	27	39	30

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	11	26	39	44
R0371	26	34	39	29
S4749	23	39	26	39
T1483	30	25	31	16
Mean	23	31	34	32

TABLE A 37

CREATININE BEFORE AND AFTER ACTH ADMINISTRATION

(mg/hr)

Subjects, Control Grp	A		A'	
	Before	After	Before	After
H9329	82.1	64.4	-	-
H8421	66.7	64.7	61.7	62.1
H3136	73.7	56.9	75.9	49.0
M7371	72.0	72.0	59.0	40.4
W4130	78.8	78.8	72.8	73.8
W7018	91.8	49.5	-	-
S 1495	72.5	34.9	60.5	62.8
W1009	63.7	35.7	78.8	32.1
Mean	75.2	57.1	68.1	53.4

Subjects, Attacking Co	B		C		D	
	Before	After	Before	After	Before	After
H4808	102.9	50.8	78.1	77.5	108.3	83.0
M0305	62.6	41.4	55.6	49.0	57.4	64.7
S4171	72.7	59.2	68.7	74.0	72.0	88.8
W1083	151.1	71.9	70.0	55.0	79.9	55.2
Mean	97.3	55.8	68.1	63.9	79.4	72.9

Subjects, Defending Co	B		C	
	Before	After	Before	After
F8576	31.3	69.5	83.6	80.0
R0371	64.0	52.4	91.5	75.0
S4749	87.5	87.8	89.8	107.5
T1483	65.6	49.0	74.2	62.4
Mean	62.1	64.7	84.8	81.2

Group Comparison

Three main groups of infantrymen were studied who were in combat of different intensities and for different periods of time. These groups are described in detail in the main body of this memorandum. Briefly, the Attacking Company spent 18 hours in intense combat and suffered 61 percent casualties; the Defending Company occupied the same position for 5 days, under less severe conditions, and took 17 percent casualties; the Control Group occupied blocking positions on the MLR in a relatively quiet area.

TABLE A38

COMPARISON OF MEAN VALUES OF URINARY AND BLOOD CONSTITUENTS OF CONTROL GROUP WITH LABORATORY DATA ON NORMAL SUBJECTS

Measure	Control Grp[a]	N	Normal subjects, US[b]
17 KS, mg/hr	0.64 (σ_M = ± .042)	24	0.695 (σ_M = ± .032)[c]
Creatinine, mg/hr	80	24	75[d]
Urinary, Na/K	3.07	24	1.93[d]
Uric Acid, mg/hr	38	24	31[d]
Eosinophils, per cu mm	235	23	248[d]
Lymphocytes, per cu mm	2904	24	3068[d]

[a]Age for Control Group ranged from 18 to 37 with a mean of 23 years.

[b]Age of all normal subjects ranged from 20 to 39 with the exception of 17 KS subjects (20-29) and eosinophil subjects (40-60).

[c]G. Pincus, L. Romanoff, and J. Carlo, "Excretion of Urinary Steroids by Men and Women of Various Ages," *J. Gerant*, 9:113 (1954).

[d]H. Hoagland, G. Pincus, F. Elmadjian, L. Romanoff, H. Freeman, J. Hope, J. Ballan, A. Berkeley, and J. Carlo, "Study of Adrenocortical Physiology in Normal and Schizophrenic Men," *A.M.A. Archives Neur. & Psychiat.*, 69:470 (1953).

It is of interest then to describe the various physiological responses of these groups; to discover whether short-term, intense combat (Attacking Company) differed markedly from long-term, less intense combat (Defending Company) in its physiological effects and to determine in turn how these two stress groups differed from the Control Group.

The Control Group's physiological data compare very closely with that obtained in laboratory work on normal subjects in the United States. Table A38 compares mean values of urinary and blood constituents of the controls with laboratory data on normal subjects. There is marked agreement in spite of the expected differences due to diet, time of collection, and general living conditions. Thus it can be stated that soldiers in blocking positions some 200 yards behind the MLR react normally on all the physiological measures.

Following combat the Attacking and Defending Companies differed in the majority of the physiological measures. These differences and their interpretation will be discussed in this section. There were also some apparent group differences in the length of recovery time which will be discussed in a later section.

White Blood Cell Measures. The Attacking Company showed an eosinopenia following combat (B) whereas the count in the Defending Company was close to normal on B but dropped 11 days following removal from combat (see section "Eosinophils"). The lymphocytes in the Attacking Company were not significantly lowered following combat until 5 days after removal from action but had not returned to normal after 22 days. On the other hand, there was a clear lymphopenia in the Defending Company after combat and recovery was not complete 11 days later (see section "Lymphocytes"). A marked shift to the left (appearance of immature neutrophils) was observed in the Attacking Company at B. Furthermore, in spite of this shift to the left a leucopenia observed in this group was due mainly to a decrease in neutrophils. The Defending Company at B showed a normal white cell count which dropped significantly at C. Also in the Defending Company the neutrophils did not show such a marked shift to the left (see section "White Blood Count").

Urinary Adrenal-Pituitary Indices. The Attacking and Defending Companies also showed different responsiveness of the adrenal gland. When several of the main indicators are studied, e.g., 17 KS, P.S. chromogens, Na/K ratios, urinary urea, and uric acid, the distinction between the two groups becomes apparent.

The Attacking Company showed a high 17 KS output, a slightly high P.S. chromogen output, a low Na/K ratio, and a high urea and uric acid excretion following exposure to combat. All these indices are complementary and indicate that the adrenal cortex was active, that it was being stimulated by the stress encountered and was secreting a larger than normal amount of adrenal hormones; and, along with this, that there was protein catabolism and a shift in the electrolyte balance.

The converse was true in the Defending Company, the less intense, long-term combat group. Here the 17 KS and P.S. chromogen excretion was below normal, the Na/K ratio was high, and urea and uric acid excretion were both in the normal range. These measures indicate that the adrenal was not responding to the stress, that it was excreting a lower than normal amount of hormones, that there was no protein catabolism, and there was an opposite shift in the electrolyte balance. Thus there appears to be a stimulation of adrenal cortical function in short-term, intense combat and a dulling of the adrenal cortical function in the long-term, less intense stress situation.

In Fig. A9 the poststress (B) Na/K ratio is plotted against the 17 KS (B) measures for both the Attacking and Defending Companies. The correlation (Pearson product moment) coefficient for this scatter is −.44, significant at the 5 percent level. In the Attacking Company those who had low 17 KS had a high Na/K ratio and conversely those with high 17 KS had low Na/K. The Defending Company does not have the range of the Attacking Company and the values are clustered around low 17 KS and high Na/K.

Figure A10 is plotted in the same manner using B urea vs B 17 KS. The correlation is .86, significant beyond the 1 percent level. Here again the Attacking Company shows a considerable spread with high 17 KS and high urea and low 17 KS and low urea. On the other hand the Defending Company data are clustered around low 17 KS and low urea values.

In Fig. A11, where 17 KS is plotted against uric acid, the same picture appears as in Fig. A10. The correlation here is .84, significant at better than

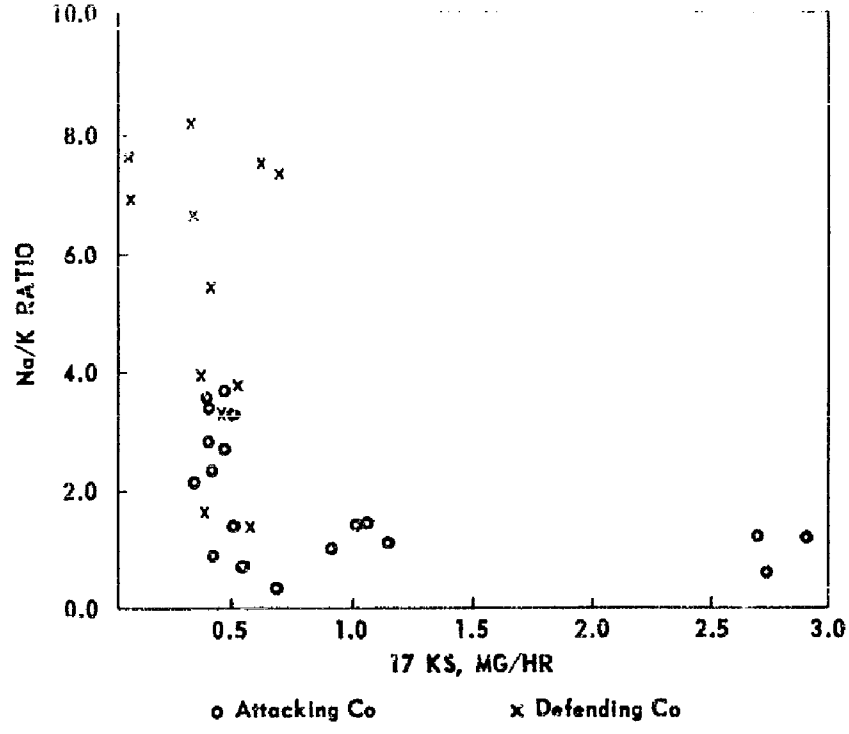


Fig. A9—Sodium/Potassium vs 17 KS, 12 Hours Poststress (Measurement B)

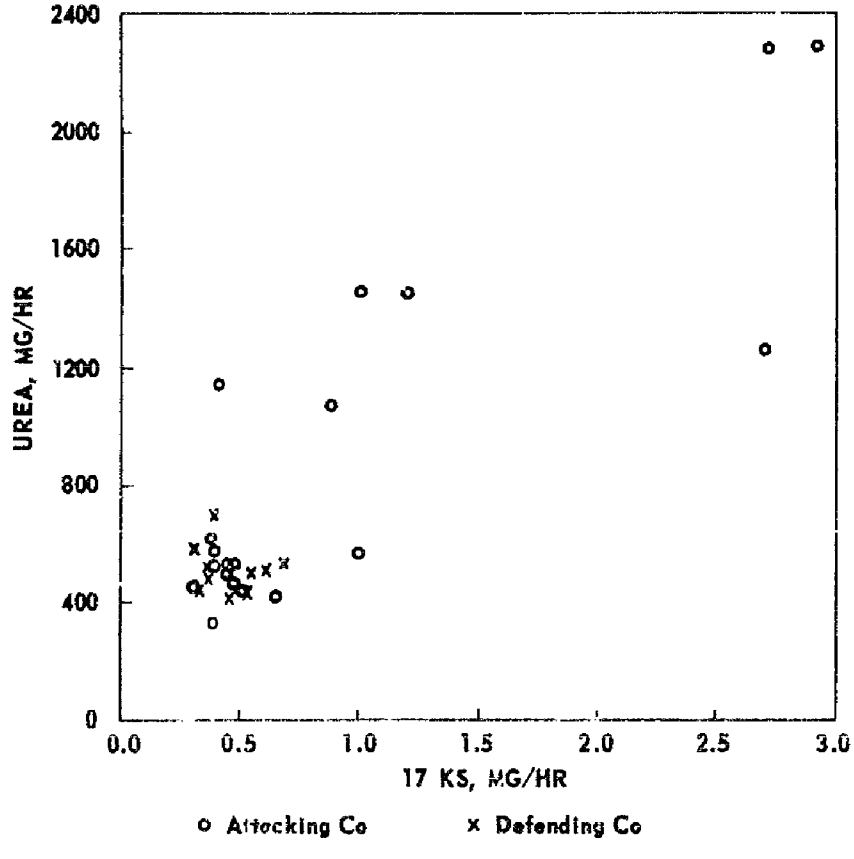


Fig. A10—Urea vs 17 KS, 12 Hours Poststress (Measurement B)

the 1 percent level.* In general, Figs. A9 to A11 show that in the Attacking Company there was increased adrenocortical activity, nitrogen catabolism, and a shift in the electrolyte balance. This was not apparent in the Defending Company where there was no increase in 17 KS and the nitrogen metabolism was in

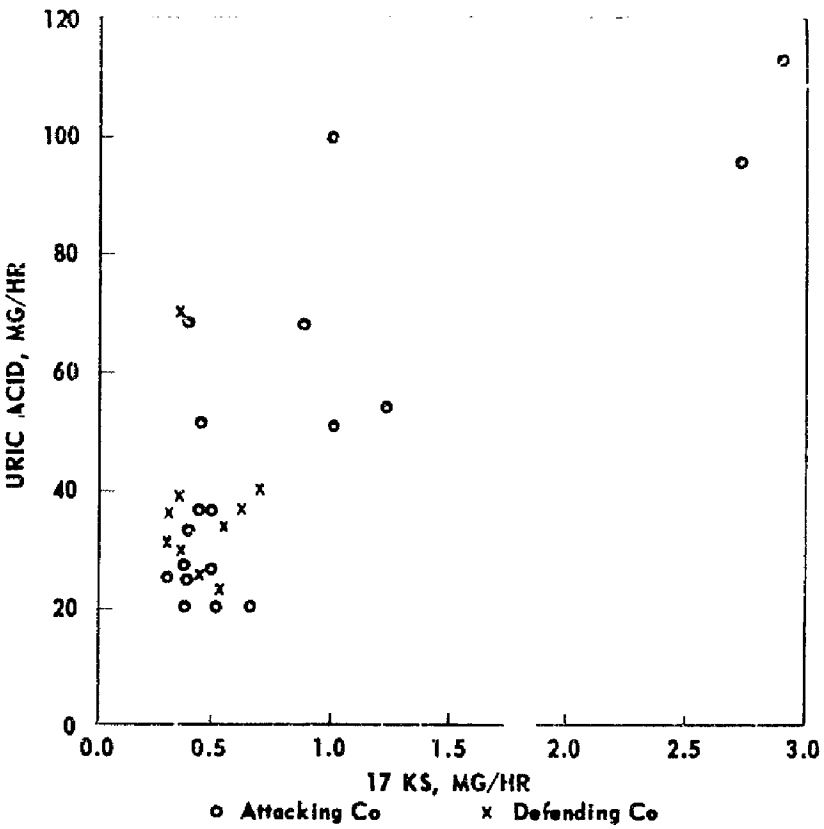


Fig. A11—Uric Acid vs 17 KS, 12 Hours Poststress (Measurement B)

the normal range. It should be noted, however, that the response of the Attacking Company was heterogeneous and that of the Defending Company homogeneous. (This particular point is discussed in the section "Individual Differences.")

The ACTH data lend some support to this distinction between the two groups although there are some unexplained changes which occur in response to ACTH, particularly in the nitrogen and sodium and potassium data. The men of the Attacking Company showed increases of 17 KS following ACTH in the post-combat measure (B). The Defending Company subjects, on the other hand, demonstrated a decline of 17 KS output following ACTH on the B measure in spite of the known stimulating action of ACTH on the adrenal gland. Eleven days later when ACTH was injected there was an increase in 17 KS excretion. The P.S. chromogen determination following ACTH provided further evidence for distinguishing between the two stress groups. In the Attacking Company the adrenal responded to ACTH as noted by the Na/K ratio indicating hyperadrenal

*These strong correlations of the various indices of adrenal function are especially noteworthy since previous experimentation has failed to reveal such meaningful relations. This may be due to the fact that the combat groups were subjected to stress much more prolonged and severe than the subjects of laboratory experimentation.[3(33)]

activity. This is in line with the results of the 17 KS and P.S. chromogens. Contrary to the 17 KS and P.S. chromogen data indicating post-ACTH hypoadrenal activity, the Na/K ratio decreased in the Defending Company in response to ACTH, which provides some evidence of adrenal activity. This would indicate that the adrenal was secreting a hormone in response to ACTH which may be specific to the electrolyte balance. This hormone may be the recently discovered electrocortin which is specific to electrolytes. Electrocortin would not affect P.S. chromogens and 17 KS but would result in an increase in Na/K ratio.[34]

TABLE A39

GENERALIZED SUMMARY OF DIFFERENCES IN PHYSIOLOGICAL DATA

Measure	Control data	Attacking Co[a]	Defending Co[a]
Blood			
Total white count	8800/cu mm	–	n
Eosinophils	235/cu mm	–	n
Lymphocytes	2900/cu mm	n	–
Urine			
17 KS	0.64 mg/hr	+	–
Na/K	3.07	–	+
Urea	500 mg/hr	+	n
Uric acid	38 mg/hr	+	n

[a]–, significantly low; +, significantly high; n, normal range

In summary the comparison of the biochemical profile of soldiers subjected to an intense battle situation (Attacking Company) vs those subjected to a less intense battle situation for a longer period of time (Defending Company) shows that the two situations have different physiological effects. Whereas in the short-term, intense stress there was an indication of increased steroid output with increased protein catabolism, in the long-term stress there was a dulling of the adrenocortical function as evidenced by the 17 KS and the P.S. chromogens and no protein catabolism. In addition the shifts in electrolyte balance were in the opposite direction for the two groups. Table A39 summarizes the biochemical profiles of the groups studied. However, it is clear from the electrolyte data observed in the Defending Company after ACTH that the adrenal was not completely nonresponsive; it was nonresponsive as far as 17 KS and P.S. chromogens were concerned, but it did respond to ACTH where some hormone, possibly electrocortin, related to the electrolyte function, was stimulated. It may be inferred that whatever hormone was being stimulated after ACTH (acting on the electrolytes) in the Defending Company, it was not detectable in the 17 KS or P.S. chromogens.

From a detailed study of the literature, Selye has prepared patterns of the various physiological responses to stress.[5(277-80)] Three such curves are shown in Fig. 5 in the main body. Each curve is divided into three phases—the alarm, resistance, and exhaustion phase. This triphasic response to stress Selye calls the "General Adaptation Syndrome." These patterns may be interpreted as reflecting increased secretion of adrenal hormones during the early stages of

stress (alarm phase) followed by a much longer period of below-normal adrenal activity as the stress is continued (resistance phase). This in turn is followed by a stage of exhaustion which may lead to death of the organism. It would appear from the data in this memo that, taken as a group, following combat (B) the Attacking Company was in the initial phase of this stress reaction, whereas the Defending Company had passed through this stage and reached the second or resistance phase.

Individual Responses

In addition to the distinctly different biochemical profiles for the Attacking and Defending Companies following exposure to combat, there is a difference in the amount of individual variation within each of the groups. In the Attacking Company the high 17 KS mean following combat (B) was accompanied by a large spread within the group (0.32 to 2.89 mg/hr). In the Defending Company the low mean was accompanied by a relatively narrow range (0.07 to 0.69 mg/hr), and in the Control Group the range was 0.28 to 1.34 mg/hr.*

Idealized curves may be drawn for each physiological response to show in general the pattern followed when the organism is exposed to stress. Indices of adrenal response such as 17 KS, nitrogen metabolism, and electrolyte response have been traced under various stress situations. The pattern for a physiological response is usually the same for various stresses. Browne et al.[35] present the case of a 23-year-old man who suffered fractures and whose physiological responses were observed for approximately 2 months. Following fracture there was a sharp increase in 17 KS with a high negative nitrogen balance (protein catabolism). Approximately 2 weeks later a fall-off was observed in both of these to a point where they were a little below normal. This was followed by a return to normal. A curve for 17 KS which follows a similar pattern is reported in operated cases by Forbes et al.[27] From the vast amount of information that Selye reports in his book, Stress, on the various measures of adrenal activity, he draws generalized curves for the various indices.[5(277-80)] Figure 5 in the main body of the memorandum presents three such curves. In summary, stress is normally responded to by an increased secretion of adrenal steroids, nitrogen catabolism, lowered Na/K ratio, etc., followed by a disappearance or reversal of these effects and then a gradual return to normal after the stress is withdrawn. If the stress is continued a stage of exhaustion is reached resulting in radical changes in the pattern of response.

If there is a generalized curve for physiological responses which is followed when an individual is exposed to stress, the narrow range of physiological response observed in the Defending Company would be expected. However, the wide range of responses observed in the Attacking Company on the various indices following combat (B) must be explained. Hypothesizing idealized curves for physiological indices for each individual the spread in the Attacking Company represents a difference in time of reaction.

*The F values comparing the variances of the Attacking and Defending Companies are given in Table A28 in the section "Urinary Steroids and Corticoids." These values are highly significant. To rule out the influence of the mean on the variance the coefficient of variation was computed for each group. The coefficient of variation for the Defending Company was found to be significantly lower than that for the Attacking Company for both 17 KS and Na/K ratios.

It appeared that all the individuals were subjected to a similar combat stress. It was therefore expected that all the subjects would show a similar physiological response to this stress. In the Attacking Company the men were up against intense enemy fire, they all went through a similar physical exertion, and they undoubtedly all thought that there was a good chance of their being killed or wounded. It is certain that they saw others severely wounded or killed. Possibly there were subtle differences in the meaning of the situation to the individuals, and some of the variation could be accounted for by this. However, it does not seem likely that the tremendous spread could be accounted for completely by a difference in meaning.

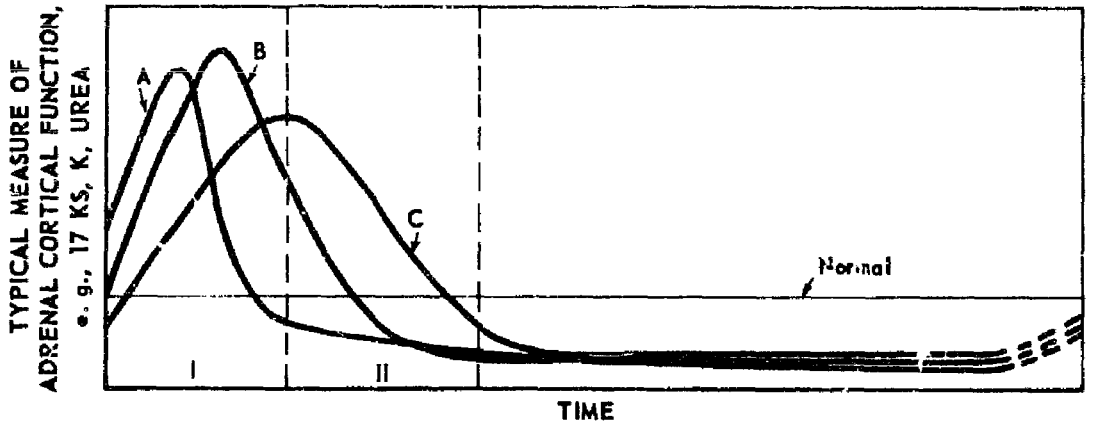


Fig. A12—Variation in Rate of Physiological Pattern Shown by Three Individuals, A, B, and C, in Stress Patterns I and II

It seems logical that each individual subjected to stress follows much the same physiological pattern; however, it would also seem logical that the rate at which each individual proceeds through the pattern would be different. In other words the physiological change is approximately the same for each individual but physiological time is different. This is graphically presented in Fig. A12. Here a typical adrenocortical measure is plotted against time. If it is assumed that stress starts acting at 0 time and continues for an indefinite period of time, the hypothesized reaction of three individuals, A, B, and C are shown by the three curves. Individual A, who normally has a high adrenocortical output, responds very rapidly at the onset of stress and proceeds through the pattern much more rapidly than Individual C, whose initial adrenocortical output is lower and whose rate of change is slower. It can be seen from this graph that if measures were made of this function at Time I there would be considerable individual variation in the measurement at that time; Individual C would show high adrenocortical response, Individual A would be low or below normal, and Individual B somewhere between A and C. Measurements made at Time II would show little variability of individuals since all would have passed into the second phase of the stress pattern.

Returning to the 17 KS data, although complete curves cannot be drawn for each individual the large spread of the data with the high mean poststress value in the Attacking Company would indicate that they were measured some-

where in the region of Time I, whereas the Defending Company, with a below normal mean and narrow range, was measured at Time II, somewhere in the resistance phase.

With this hypothesis, considerable meaning can be attached to the low 17 KS values in the Attacking Company. When these individuals were subjected to stress or were in an anticipatory state their 17 KS started to rise at a more rapid rate and they went through the typical reaction faster. When they were observed following 18 hours in combat they had passed through the high part of the pattern and were approaching or were in the low phase of the

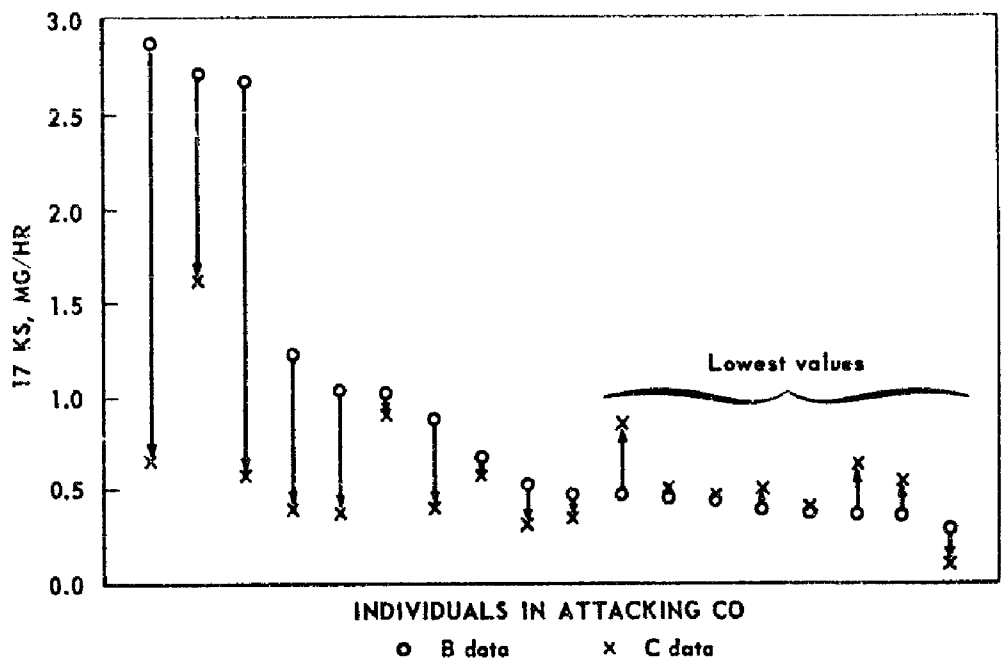


Fig. A13—Changes in 17 KS from Measures B to C, Attacking Co

pattern. Data for these individuals, then, should resemble the data on the Defending Company. Evidence for this is shown in Fig. A13. Here the postcombat (B) 17 KS data of the Attacking Company are plotted in descending order of value. Of the eight lowest values (indicated by the brace), seven of them rise on recovery (indicated by the upward direction of the arrow).* The mean uric acid, urea, and Na/K values for these eight subjects are in close agreement with the mean values in the Defending Company. The mean values for the remaining 10 subjects are considerably different from those for the Defending Company (see Table A40). The response of 44 percent of the Attacking Company is very similar to the mean response of the Defending Company, already identified as being in a more advanced stage of the stress reaction.

Figure A14 shows the 17 KS data for the Defending Company plotted in the same manner as the Attacking Company. The seven lowest subjects, or

* Since repeat data are not available on the Control Group to determine the reliability of 17 KS it may be possible that the falling of high values and the rising of the low values are due to regression to the mean. 17 KS have been reported to be highly reliable, however; each individual has a characteristic level of 17 KS excretion for both day and night.[33,36] It is therefore doubtful that the shift to the mean on C could be due to regression.

64 percent of the group (indicated by a brace) return toward normal on recovery, whereas the four highest come down to normal. However, there are no extremely high values as there are in the Attacking Company.

Using the terms described by Selye (the alarm, resistance, and exhaustion phases) to characterize the portions of the idealized pattern, after 18 hours in combat 44 percent of the men in the Attacking Company gave all indications

TABLE A40

COMPARISON OF MEASURES OF HIGH AND LOW 17 KS EXCRETERS IN ATTACKING COMPANY WITH THOSE OF DEFENDING COMPANY

Measure	Mean of high 17 KS excreters, Attacking Co (N = 10)	Mean of low 17 KS excreters, Attacking Co (N = 8)	Mean of Defending Co
17 KS mg/hr	1.41	0.41	0.42
Urea mg/hr	1168	580	487
Uric acid mg/hr	71	37	34
Na/K	1.2	2.8	5.1

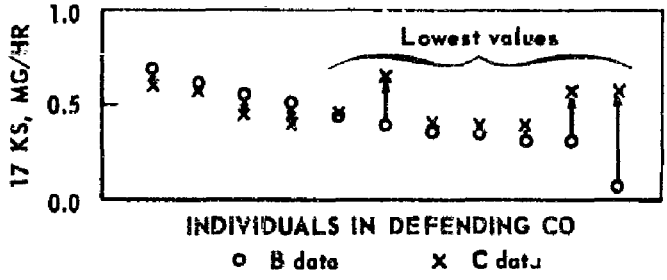


Fig. A14—Changes in 17 KS from Measures B to C, Defending Co

of having reached the resistance phase, while the remaining 56 percent were in the alarm phase. The Defending Company as a whole, after 5 days of less intense combat, was in the stage of resistance, but there was a portion of the group which was either in the last stages of the alarm phase or had entered the exhaustion stage. If there were complete ACTH data on these four highest individuals in the Defending Company it might be possible to place them in either the alarm or exhaustion phase. As it is, the ACTH test was administered to only one subject in this group of four, and he showed no increase in 17 KS excretion, indicating that the adrenal was not responding. This is contrary to the results with the Attacking Company where, when they appeared to be in the alarm phase and ACTH was administered, there was a response.

Physiological Recovery

The data obtained in Korea provide some information about the time necessary for the physiological processes to return to normal. It is important for the military commander to know whether it takes a few hours, several days,

or longer for the organism to return to normal, physiologically, after combat stress.*

It is clear from the previous sections that the Attacking and Defending Companies present different biochemical profiles. As a group, the Attacking Company shows hyperadrenocortical activity, the Defending Company hypoadrenocortical activity. The former condition is generally recognized as the normal response to stress when it is first encountered, the latter stage is reached when stress is continued over a period of time. It is important for future work in this area to determine the pattern and time of recovery for hyperadrenocortical and hypoadrenocortical activity.

In Figs. A15 to A18 the major physiological measures are plotted for the Control Group and the Attacking and Defending Companies. The mean for the Control Group is considered the normal response and forms the base line; the means for the Attacking and Defending Companies at B, C, and D measurements are percentages of this mean. The measuring times on the Attacking and Defending Companies are not the same as can readily be seen in Fig. 3 of the main body of this memorandum. Since successive measurements at frequent intervals were not made the results, so far as recovery time is concerned, can be only approximate.

Figure A15 presents the comparisons on white blood count measures. In general the blood count started to return to normal in the Attacking Company 5 days (C) after removal from combat. Twenty-two days (D) after removal from combat it was for all purposes normal. It was not possible to determine, however, when it returned to normal from the available data, but it certainly was somewhere between 5 and 22 days after removal from combat.

The mean eosinophil and white blood cell counts in the Defending Company, on the other hand, were actually lower 11 days after removal from combat (C) than 12 hours after removal (B). The lymphocytes were approaching normal 11 days following removal from combat.

As a group, then, the Attacking Company appeared to be on the way to recovery 5 days after removal from combat, whereas the Defending Company, even 11 days after removal from combat, in general did not show the approach toward normal.

Individually, the subjects in the Attacking Company showed a variety of responses.[2(23-24)] On lymphocyte counts, for example, some returned to normal on C measure, others continued to drop on C, or showed no change on C, and then on D measure, whereas some showed normal counts, others still had not returned to normal. The same general picture was true of eosinophils.

The electrolyte data in Fig. A16 present essentially the same picture. Five days after removal from combat the mean Na/K ratio in the Attacking Company crossed over the normal Na/K value, whereas in the Defending Company, even after 11 days out of combat, the mean electrolyte response had just dropped to within 12 percent of normal. To support this, the urinary chloride response in the Attacking Company had returned to near normal at C, whereas in the Defending Company there was a low output after 11 days.

*It has been pointed out previously that one of the many possible reasons that the psychological tests did not show any stress effects is this question of recovery time. Recovery may be so rapid in the areas tested or the subject may be able to recover momentarily and successfully complete the test.

The nitrogen picture in Fig. A17 shows the Attacking Company returning to normal within 5 days of leaving combat. The Defending Company was in the normal range following combat and remained there.

The 17 KS data plotted in Fig. A18 show that the Attacking Company recovered after 5 days out of combat, whereas the mean 17 KS excretion of the Defending Company was still approximately 22 percent less than the normal value 11 days after combat.

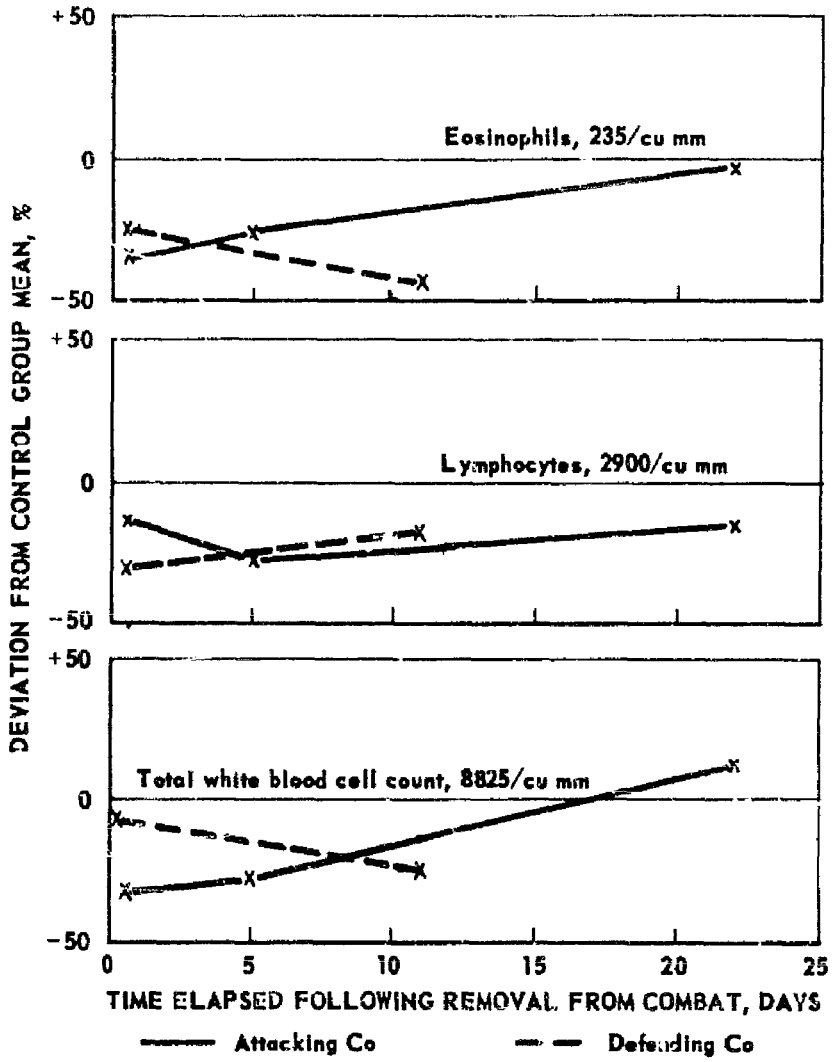


Fig. A15—Mean Blood Counts for Poststress Measures

Briefly, it appears as though the electrolyte, nitrogen, and 17 KS in the Attacking Company returned to normal levels a few days after combat. The blood counts approached within 30 percent of normal levels. On the other hand the Defending Company, after 11 days out of combat, was still low on 17 KS and blood count and above normal (10 percent) on Na/K and below normal on chloride. The nitrogen data fell within normal limits following combat and remained there in the Defending Company. It can be concluded that recovery from exposure to combat requires at least several days, rather than hours, and recovery

from hyperadrenocortical activity is more rapid than recovery from hypoadrenocortical activity. From inspection of the data, assuming a nearly linear relation between time and increments of physiological change, limits can be placed on the length of the recovery time. The Attacking Company recovered in not less than 4 days and not more than 8 days. The Defending Company recovered in not less than 11 or more than 16 days.

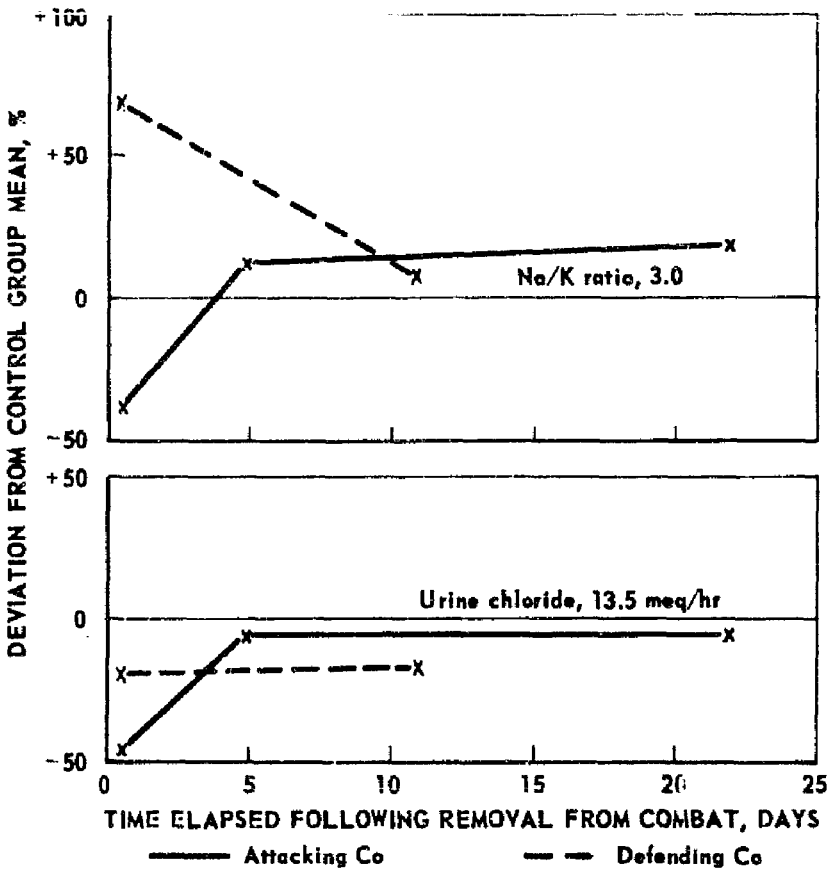


Fig. A16—Mean Electrolyte Balance for Poststress Measures

Problems of Measurement

On the basis of results obtained in this study, suggestions and recommendations for a battery of tests for a future field study of this type can be made. Detailed description of laboratory analysis and techniques for administration of the specific measures employed in this study are given in ORO-T-41(FEC).

Blood Measurements. No difficulty was encountered in taking blood samples although extra sterilization precautions had to be observed because of possible hepatitis infection. Modifications of laboratory techniques were made to facilitate field use; these are described in detail in ORO-T-41(FEC). In general the results were consistent with reports of other stress research; however, the team felt that of the two, blood or urine samples, urine was the more practical and yielded just as valuable results. A blood sample must be immediately centrifuged and the plasma drawn off prior to analysis for sodium, potas-

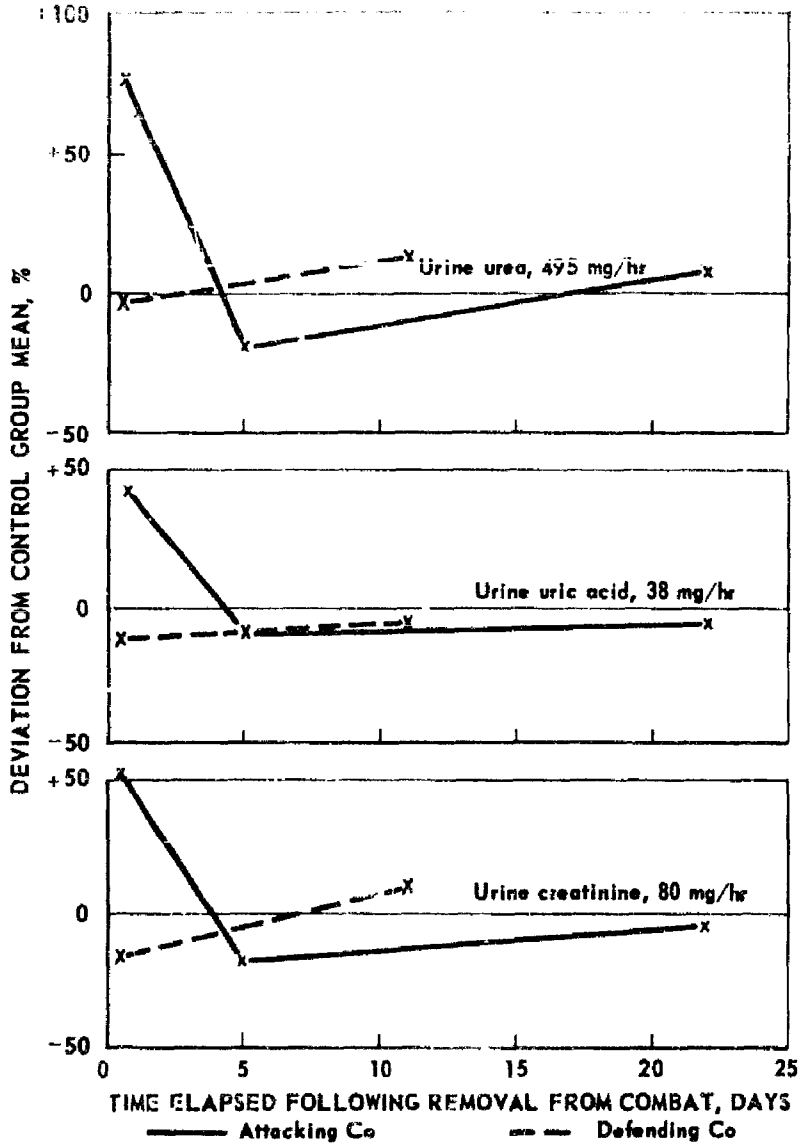


Fig. A17—Mean Nitrogen Excretion for Poststress Measures

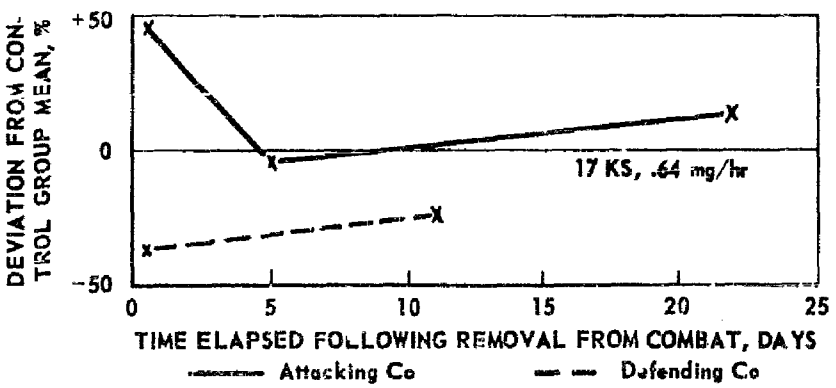


Fig. A18—17 KS Poststress Measures

sium and chloride, whereas with urine, analysis can begin at once. Preservative techniques plus the necessity of going into a vein to get a sufficiently large sample (10 to 20 cc) make the taking of blood samples more difficult.

Hematology. Differential blood counts were performed without difficulty. Diurnal variation in eosinophil counts makes it necessary to have repeated and controlled observations on the same subjects. In the analysis of this data certain subjects with blood counts out of the normal range were dropped. This was done on the basis of previous clinical data which have established that extremely high counts indicate pathological conditions, e.g., hay fever.

Hematocrit Value. Determining hematocrit values in the field is relatively easy. The hematocrit values were normal for the stressed groups; i.e., there was no detectable change as a result of combat stress.

Blood Volume. The measurement of blood volume used in this study involved a complicated procedure for preparing the radioactively tagged cells, injecting them into the subjects, withdrawing a blood sample, and then measuring their distribution in the sample. This procedure occupied the full time of one team member. Blood volume was highly variable in the controls, and the stressed groups showed no change in blood volume after combat. For this study the measurement of blood volume was helpful in verification of other data.

Plasma Chloride, Sodium and Potassium. The stressed groups showed no change in plasma chloride following combat. There was a breakdown in the blood cells which affected the potassium values. Any value of potassium higher than 6.5 meq/l was unreasonable and was eliminated as an error of collection. It is difficult to obtain and prepare the sample for the study of blood electrolytes, and the fact that the blood constituents are fairly constant means that highly accurate analytical methods are necessary to detect changes. Therefore plasma electrolyte data were not used to assess the effect of combat stress on the groups.

Plasma Carbon Dioxide. The method used was satisfactory but time consuming. There was a slight lowering of CO_2 in the plasma prior to combat; however, the results showed no effect of combat.

Plasma Total Cholesterol. The method used proved satisfactory for field conditions. It is necessary to measure cholesterol within 24 hours after collecting the sample, for otherwise the cholesterol level will fall appreciably. As pointed out earlier, reliable data were obtained; however, complete knowledge as to the meaning of the data must await further experimentation.

Blood Sugar. Blood sugar showed no specific changes as a result of combat. Again, although the technique was satisfactory in the field situation, this measure was not sensitive to stress.

Blood Urea. The measure of blood urea may be used to verify the urine nitrogen values. The blood urea values confirmed the nitrogen catabolism in the Attacking Company.

Mecholyl Test. Mecholyl was used as a possible means of measuring the reactivity of the autonomic nervous system, especially those portions concerned with vascular and cardiac adjustments. Mecholyl chloride was injected and blood pressure and heart-rate measures taken before, during, and after injection. The curves were reproducible in control subjects and did show effects of stress in the Defending Company. Although no laboratory analysis is required the test is not practical because of the discomfort to the subjects

and the necessity for the subject to be inactive for a period of about 30 minutes. It is recommended that in acute stress a simple measure of the blood pressure on the resting subject may be as useful an index of autonomic tonus as the more elaborate Mecholyl test. Blood pressure could be obtained on more subjects than the Mecholyl test and would not involve either the discomfort or the long period of inactivity.

Saliva Measurement. Conceivably, obtaining samples of saliva and examining the samples for various excretory products would be one of the easiest ways to evaluate the effects of stress. The means of collection, however, gives rise to many variables which can contaminate the saliva—food in the teeth, amount of saliva, etc. In this study saliva was analyzed for sodium and potassium content. Neither was found to be a reliable index in the Control Group subjects. Further experimentation with this technique may prove that it is simpler than either urine or blood.

Urine Measurement. Perhaps the most satisfactory sampling from the standpoint of practicality, simplicity, and ease of preservation was urine. The subject was asked to void, and then during the test period, 2 to 3 hour, or overnight in the case of ACTH testing, to collect all urine in a bottle. The subjects were very cooperative and collection times were considered fairly accurate.

Urinary Chloride. Urine volume changes are reflected in urinary chloride. If water is taken then total chloride increases, distorting the picture; therefore the Na/K ratio is preferred. Chloride excretion is very similar to sodium and although urinary total chloride determination is a relatively simple method, there should be either a reliable collection time or creatinine values. Because it is supplementary to the Na/K ratio (see below), it is not felt that this measure is necessary.

Urinary Sodium and Potassium. One of the most satisfactory measures made by the team was the Na/K ratio. Using a flame photometer, sodium and potassium values for urine can be determined without the necessity of accurate collection times or care in restricting water intake. The fact that sodium and potassium values are expressed as a ratio makes it unnecessary to take into account large variations in absolute levels.

Urine Nitrogen. Urine urea, uric acid, and creatinine reflect nitrogen metabolism. Creatinine excretion has been considered to be fairly constant, and therefore urine constituents are normally expressed in relation to milligrams of creatinine per 100 cc of urine, rather than milligrams per hour, which depends on accurate knowledge of collection times. The mean output of creatinine has been observed to increase with stress;[30] such was the case in this study, and as a result the nitrogen and other urine data, which were originally expressed in terms of creatinine, were reanalyzed and expressed in terms of milligrams per hour. This was done after the specific gravity and other values had been examined to ascertain whether the collection times were sufficiently accurate.

Urine Glucose. In general, laboratory studies have shown an increase in urine glucose following stress, ACTH, etc. Although changes were observed under stress in this study the results for urinary sugar are not clear-cut and interpretation is difficult at this time. Since carbohydrate metabolism is very important for the organism during a period of stress this measure may take on added importance as additional research is accomplished.

17 Ketosteroids. The method of extracting the urine samples to dryness in Korea and shipping these to the United States for complete analysis proved entirely satisfactory. Although the expense involved in 17 KS determination is considerable in comparison with other analyses, the information supplied justified the expenditure. If in future work it were desired to study large numbers of subjects it would be more practical, from the point of view of cost and effort expenditure, to make 17 KS determinations on only a sample of the total group.

P.S. Chromogens. The method of preparing the samples in Korea and shipping them to the United States for analysis proved feasible for this measure also. The Porter-Silber data are scattered because only a portion of the total samples extracted were analyzable. In all probability this scatter is due to the high blank, which makes readings impossible, and to other defects in the particular method used, as well as to possible technical errors in preparation for analysis. At present there is a method described by Glenn and Nelson[37] which does not have this difficulty of analysis, and the high blanks have been obviated. Although these determinations are valuable in assessing the hormonal reserve and excretion of the adrenal cortex, the expenditure of cost and effort should be a consideration in subsequent work.

ACTH Test. The injection of 2.0 ml ACTH in gel intramuscularly was accomplished without any difficulty for the subjects. Since ACTH does have some generalized effects, caution must be observed in its administration. The effect on steroid output, electrolytes, and carbohydrate and nitrogen metabolism in the urine was measured, and the above comments on analysis of these measures without ACTH apply. In general the method was satisfactory and contributed valuable data to the interpretation of the adrenal-pituitary function under stress.

Annex 1

ADDITIONAL RAW DATA

These tables provide raw data supplementary to those found in ORO-T-41 (FEC).

TABLE A41

17 KS AND P.S. CHROMOGEN MEASUREMENTS

Part A. Control Group

Subjects	17 KS, mg/hr	P.S. chromogens, mg/hr	
	A	A	A'
A5075	0.28	—	—
C5107	0.68	—	—
H9329	0.55	—	—
after ACTH	[illegible]	—	—
H8421	0.42	—	—
after ACTH	0.46	1.5	—
H3136	0.72	—	0.32
after ACTH	0.68	0.92	—
K3195	0.51	—	—
M3285	1.34	—	—
M7371	0.59	—	0.27
after ACTH	0.74	—	—
S6941	0.52	—	—
W4130	0.63	—	0.43
after ACTH	0.28	—	—
W9893	0.72	—	—
W7018	0.89	0.28	—
after ACTH	0.41	0.64	—
B9350	0.44	—	—
B8787	0.58	—	—
C9356	0.59	—	—
C6378	0.72	—	—
F0536	0.57	—	—
G2474	0.8[illegible]	—	—
G6497	[illegible].32	—	—
K4360	0.49	—	—
M5261	0.70	—	—
O7035	0.54	—	—
S1495	0.65	0.25	—
after ACTH	0.23	—	—
W1009	0.64	—	—
after ACTH	0.28	—	—

TABLE A41 (continued)

Part B. Attacking Company

Subjects	17 KS, mg/hr				P.S. chromogens, mg/hr			
	A	B	C	D	A	B	C	D
A8537	0.74	—	—	—	0.33	—	—	—
C4136	1.05	1.04	0.37	0.59	—	0.32	—	—
C5898	0.78	—	—	—	0.29	—	—	—
E3096	0.88	—	—	—	—	—	—	—
G5761[a]	0.61	—	—	—	0.27	—	—	—
P1061[a]	0.81	—	—	—	—	—	—	—
Schow[a]	1.03	0.51	—	—	—	0.05	—	—
Smith[a]	1.76	—	—	—	—	—	—	—
S5171	0.49	—	—	—	—	—	—	—
A4492	0.39	—	—	—	—	—	—	—
B7531	0.80	0.39	0.65	—	—	—	0.67	—
H7321	0.67	0.48	0.86	0.74	0.40	—	0.42	0.84
J4852[a]	0.78	—	—	—	0.57	—	—	—
M3290	0.43	—	—	—	—	—	—	—
O4697	0.40	—	—	—	—	—	—	—
R2423	0.13	—	—	—	—	—	—	—
B2003	—	—	—	—	—	—	—	—
H4808	—	1.01	0.93	1.22	—	0.34	0.88	0.26
after ACTH	—	1.75	0.79	—	—	0.37	—	1.40
J0703	—	—	—	—	—	—	—	—
M7579	0.51	—	—	—	—	—	—	—
M1604	0.37	—	—	—	—	—	—	—
M0305	—	0.32	0.13	0.38	—	0.13	0.41	0.45
after ACTH	—	0.39	0.51	—	—	0.25	—	0.78
Moss	0.69	—	—	—	—	—	—	—
W3173	0.40	—	—	—	0.14	—	—	-
A8639	—	0.46	0.52	0.85	—	—	—	—
H1864	—	0.66	0.59	0.98	—	—	—	0.72
H1296	—	0.78	1.19	0.56	—	—	—	0.37
J4226	—	0.38	0.56	0.40	—	—	—	—
J8455	—	0.39	0.41	—	—	—	—	—
L6206	—	0.49	0.36	—	—	—	—	—
M9987	—	0.42	0.51	0.67	—	—	0.35	—
P4185	—	2.89	0.65	1.03	—	0.96	0.37	0.34
S7808	—	1.23	0.39	—	—	—	0.38	—
S4171	—	0.45	0.48	0.70	—	—	0.60	—
after ACTH	—	0.59	0.75	—	—	0.50	—	0.9
S4917	—	2.68	0.59	0.55	—	—	0.65	—
T9985	—	0.51	0.33	—	—	0.81	0.13	—
W0218	—	0.39	—	—	—	—	—	—
W2990	—	2.72	1.62	—	—	2.30	0.85	—
W1083	—	0.88	0.42	0.65	—	0.19	0.26	0.47
after ACTH	—	0.73	0.42	—	—	—	—	0.74

[a]Subjects who were wounded.

TABLE A41 (continued)

Part C. Defending Company

Subjects	17 KS, mg/hr		P.S. chromogens, mg/hr	
	B	C	B	C
F8576	—	0.63	—	1.1
after ACTH	0.42	0.77	—	—
B5392	0.37	0.42	0.30	—
H4368	0.40	0.67	—	—
J4476	0.61	0.58	0.24	—
L0624	0.69	0.59	0.82	1.0
M2830	0.36	0.39	—	—
O8834	0.07	—	—	—
P4431	0.55	0.45	—	—
P0411	0.33	0.41	0.29	—
R0371	0.45	0.46	—	0.92
after ACTH	0.26	0.48	—	1.12
S4749	0.52	0.40	0.45	0.29
after ACTH	0.33	0.51	0.39	1.20
T1483	0.32	0.59	0.08	0.29
after ACTH	0.16	0.69	0.024	1.71
W7605	0.07	0.59	0.02	0.56
after ACTH	0.16	0.61	0.16	0.83

Part D. Psychiatric Casualties[a]

Subjects	17 KS, mg/hr		P.S. chromogens, mg/hr	
	1st measurement	2d measurement	1st measurement	2d measurement
W4352	0.38	0.48	—	0.33
after ACTH	0.61	0.50	0.52	4.40
P4360	0.25	—	0.37	0.144
after ACTH	0.78	1.24	—	3.40
S7571	0.48	1.18	—	0.14
after ACTH	0.47	—	—	—
G0442	0.61	—	—	—
after ACTH	1.09	—	—	—
P5803	(no urine sample taken)			

[a] This group appeared as Group VII in ORO-T-41 (FEC). The data are presented here since they are additional to that in ORO-T-41 (FEC) but no analyses or interpretation has been made in this memorandum. ORO-T-41 (FEC) should be referred to for a description of the cases and the exact times for first and second measurement.

TABLE A 42

SODIUM AND POTASSIUM MEASURES IN URINE, SALIVA, AND PLASMA
(meq/l)

Part A. Control Group

Subjects	Urine				Saliva				Plasma			
	Sodium		Potassium		Sodium		Potassium		Sodium		Potassium	
	A	A'	A	A'	A	A'	A	A'	A	A'	A	A'
A5075	87.7	68.4	29.5	46.6	41.0	13.40	21.8	22.0	144	134.5	4.4	4.4
C5107	152.8	232.0	51.6	65.4	22.5	9.0	23.6	21.5	142	135.4	4.5	4.6
H9329	227.5	—	64.1	—	39.8	—	20.9	—	146	—	4.2	—
after ACTH	102.4	—	78.8	—	—	—	—	—	—	—	—	—
H8421	230.3	164.2	79.3	52.6	29.0	10.5	28.2	23.2	145	137.6	4.2	3.9
after ACTH	84.8	45.7	108.2	50.0	—	—	—	—	—	—	—	—
H3136	254.8	232.0	68.0	53.8	39.8	18.8	19.5	19.7	149	159.0	4.6	3.4
after ACTH	93.5	71.7	114.8	44.2	—	—	—	—	—	—	—	—
K3195	162.1	—	49.0	—	33.6	—	17.6	—	142	—	4.3	—
M3285	282.7	277.2	100.0	67.6	41.0	12.7	17.9	17.1	152	136.8	4.2	3.6
M73 '1	172.4	171.1	61.6	80.4	46.6	11.2	22.8	25.6	143	138.6	4.9	5.1
after ACTH	134.8	70.7	71.2	46 6	—	—	—	—	—	—	—	—
S6942	125.0	156.0	33.3	59.0	29.0	14.8	21.7	22.8	146	136.7	4.4	3.6
W4130	250.0	199.9	97.5	73.4	25.8	10.1	22.6	27.1	146	138.5	4.6	4.6
after ACTH	121.7	159.3	81.7	122.4	—	—	—	—	—	—	—	—
W9893	224.1	—	62.8	—	28.0	—	22.6	—	139	—	4.5	—
W7018	198.8	210.8	53.0	75.3	12.2	4.8	19.6	19.2	142	140.1	3.9	3.4
after ACTH	42.4	—	54.1	—	—	—	—	—	—	—	—	—
B9350	89.2	43.5	39.8	27.6	25.2	7.6	20.2	21.5	150	136.8	4.2	3.9
B8737	234.6	128.4	73.7	29.9	10.0	11.6	22.5	24.6	146	146.0	4.3	7.0
C9358	227.2	84.9	84.6	33.6	36.0	21.5	18.6	16.5	144	133.1	4.2	3.1
C6378	258.5	248.3	105.7	69.6	21.6	12.7	28.4	24.5	144.1	144.0	7.2	4.0
F0536	243.2	200.3	99.6	88.1	16.6	4.0	19.4	29.1	145	139.4	4.1	3.7
G2474	182.5	—	42.7	—	9.2	—	18.6	—	143.7	—	3.7	—
G6497	234.8	—	51.3	—	17.0	—	19.1	—	146.0	—	2.9	—
K4350	172.4	—	53.4	—	6.2	—	23.0	—	142	—	3.9	—
M5261	159.8	219.8	42.3	83.2	42.5	21.5	20.8	23.2	138.3	141.3	2.6	3.7
O7035	100.6	160.2	60.9	83.0	29.0	10.9	20.3	21.8	147.5	138.1	3.8	5.7
S1495	164.1	77.1	59.9	25.6	37.4	13.8	20.4	20.8	148	134.5	3.0	3.8
after ACTH	34.8	50.0	35.0	32.3	—	—	—	—	—	—	—	—
W1009	171.0	89.8	97.4	53.8	7.4	10.9	20.8	33.3	138	143.9	5.2	5.3
after ACTH	104.7	84.0	77.2	87.9	—	—	—	—	—	—	—	—

TABLE A42 (continued)

Part B. Attacking Company (Saliva Measure Excluded for This Group)

Subjects	Urine								Plasma							
	Sodium				Potassium				Sodium				Potassium			
	A	B	C	D	A	B	C	D	A	B	C	D	A	B	C	D
A8537	128	—	—	—	106	—	—	—	125.3	—	—	—	3.7	—	—	—
C4136	185	67	220	244.9	103	49	37	59.4	139	150	139	142.5	6.6	3.5	3.4	4.6
C5898	189	—	—	—	99	—	—	—	136	—	—	—	9.6	—	—	—
E3096	179	—	—	—	102	—	—	—	137	—	—	—	7.7	—	—	—
G5761[a]	175	—	—	—	99	—	—	—	124	136	—	—	7.2	4.3	—	—
P1061[a]	156	—	—	—	77	—	—	—	146	153.7	—	—	9.8	4.2	—	—
Schow[a]	98	108	—	—	110	145	—	—	138	151.3	—	—	6.4	5.2	—	—
Smith[a]	198	—	—	—	94	—	—	—	—	146.5	—	—	—	4.8	—	—
S5171	171	—	—	—	46	—	—	—	131	—	—	—	6.5	—	—	—
A4492	215	—	—	—	72	—	—	—	154.7	—	—	—	6.9	—	—	—
B7531	156	117	217	160.2	107	50	68	99.3	143	132	156	139.4	6.6	3.5	4.0	5.2
H7321	161	90	205	177.2	93	62	54	112.3	145.6	148	137	166.0	10.6	4.0	3.7	4.1
J4852[a]	185	—	—	—	—	50	—	—	132	153.5	—	—	7.0	5.3	—	—
M3290	136	—	—	—	54	—	—	—	163.5	—	—	—	10.6	—	—	—
O4697	166	—	—	—	108	—	—	—	—	—	—	—	—	—	—	—
R2423	208	—	—	—	61	—	—	—	—	—	—	—	—	—	—	—
B2003	168	—	—	—	57	—	—	—	133.7	—	—	—	8.7	—	—	—
H4808	121	89	302	227.2	92	63	62	57.9	147	163.7	145	159.2	6.9	7.7	3.9	5.8
after ACTH	—	24	122	92.4	—	65	74	88.0	—	—	—	—	—	—	—	—
J0703	138	—	—	—	100	—	—	—	—	—	—	—	—	—	—	—
M7579	84	—	—	—	96	—	—	—	140	—	—	—	7.1	—	—	—
M1604	176	—	—	—	109	—	—	—	134	—	—	—	6.2	—	—	—
M0305	202	111	170	217.4	83	54	59	60.3	140.7	143	142	163.5	8.1	3.9	4.0	6.0
after ACTH	—	65	71	168.1	—	44	34	88.1	—	—	—	—	—	—	—	—
Moss	120	—	—	—	99	—	—	—	141.2	—	—	—	10.1	—	—	—
W3173	185	—	—	—	100	—	—	—	—	—	—	—	—	—	—	—
A8639	—	226	200	231.3	—	261	95	56.7	—	139	143.5	167.2	—	4.1	4.5	4.5
H1864	—	17	131	126.8	—	38	36	38.5	—	167.0	142	139.5	—	3.5	4.4	3.2
H1296	—	42	117	172.7	—	8	68	57.9	—	150.9	140	158.5	—	6.1	4.0	2.8
J4426	—	183	199	269.9	—	51	45	35.9	—	150	139	146.0	—	3.6	4.1	4.5
J8455	—	172	278	—	—	50	58	—	—	140	129	—	—	3.9	5.0	—
L6206	—	64	278	—	—	19	50	—	—	134.8	146	—	—	8.2	3.7	—
M9987	—	213	214	249.2	—	92	68	62.8	—	142.1	140	159.6	—	4.5	4.5	7.8
P4185	—	76	105	134.7	—	63	36	35.5	—	147	140.7	143.9	—	3.5	4.5	4.6
S7808	—	58	176	—	—	51	78	—	—	133	141	—	—	3.4	3.7	—
S4171	—	153	195	170.2	—	58	69	60.3	—	136	143	148.0	—	3.7	3.7	6.2
after ACTH	—	62	99	189.1	—	90	139	104.5	—	—	—	—	—	—	—	—
S4917	—	78	147	80.4	—	68	111	99.6	—	135	138	165.5	—	3.8	3.8	2.4
T9985	—	38	213	254.3	—	54	44	47.7	—	138	138	137.3	—	2.8	4.1	3.7
W0218	—	40	179	276.2	—	51	73	88.1	—	143.9	145	137.2	—	5.0	3.9	5.3
W2990	—	47	180	260.4	—	84	62	41.9	—	138	150	135.9	—	4.0	7.1	2.9
W1083	—	53	175	227.9	—	54	38	50.4	—	155	136	151.7	—	3.6	7.2	4.6
after ACTH	—	84	170	177.6	—	59	86	95.6	—	—	—	—	—	—	—	—

[a] Subjects who were wounded.

TABLE A42 (continued)

Part C. Defending Company

Subjects	Urine				Saliva				Plasma			
	Sodium		Potassium		Sodium		Potassium		Sodium		Potassium	
	B	C	B	C	B	C	B	C	B	C	B	C
F8576	163	360.4	53	43.0	10.9	—	17.0	—	140	142.6	4.3	6.8
after ACTH	135	134.8	78	56.2	—	—	—	—	—	—	—	—
B5392	107	50.5	63	70.2	—	14.7	—	21.5	133	139.0	4.7	3.7
H4568	391	195.5	72	62.4	21.4	14.9	19.2	19.0	146.5	134.0	6.5	4.6
J4476	351	206.1	47	51.6	9.6	10.9	22.7	20.8	146	154.2	4.3	6.1
L0624	231	197.8	31	73.1	7.3	11.2	19.8	21.8	139.7	151.2	8.8	6.9
M2880	189	224.0	49	91.1	40.0	38.6	12.8	19.1	162	166.6	3.4	6.8
O8834	159	—	21	—	10.5	—	19.7	—	140.2	—	4.7	—
P4431	96	218.8	71	59.4	10.7	10.9	22.3	22.9	132	141.3	5.3	4.3
P0411	109	143.4	16	41.0	23.2	26.8	16.8	21.8	143.7	150.8	4.2	4.0
R0371	143	191.1	43	75.6	10.7	24.2	17.6	16.8	148	137.6	3.9	3.3
after ACTH	41	80.4	32	114.8	—	—	—	—	—	—	—	—
S4749	180	191.3	57	53.8	15.5	13.8	17.1	16.7	141.2	162.3	4.2	7.9
after ACTH	57	97.9	42	70.2	—	—	—	—	—	—	—	—
T1483	207	77.1	25	24.1	6.4	10.9	18.6	21.5	150	145.7	4.4	4.7
after ACTH	89	70.7	44	110.5	—	—	—	—	—	—	—	—
W7805	113	75.3	16	29.5	5.9	6.9	22.7	22.9	139.0	166.7	3.7	4.6
after ACTH	51	61.4	30	67.9	—	—	—	—	—	—	—	—

Part D. Psychiatric Casualties

Subjects	Urine				Plasma			
	Sodium		Potassium		Sodium		Potassium	
	1st meas	2d meas	1st meas	2d meas	1st meas	2d meas	1st meas	2d meas
W4352	96	208	98	117	141	141	4.3[a]	6.9[a]
after ACTH	—	40	—	62	—	—	—	—
P4360	—	147	—	151	137	131	4.4[a]	6.7[a]
after ACTH	57	22	123	89	—	—	—	—
S7571	152	155	68	42	143.8	159	3.9	3.4
after ACTH	118	—	170	—	—	—	—	—
G0442	139	—	68	—	147	—	3.7	—
after ACTH	123	—	122	—	—	—	—	—
P5803	—	—	—	—	147	—	3.2	—

[a] These data are questionable because of hemolysis.

TABLE A43

NEUTROPHIL DIFFERENTIAL
(Percent of total WBC count)

Part A. Attacking Company

Subject	B measure					D measure				
	Myelocyte	Juvenile	Band	Segmented	Schilling index	Myelocyte	Juvenile	Band	Segmented	Schilling index
B7531	5	12.5	15.0	20.5	1.59/1	—	—	—	—	—
C4136	5.5	9	5.9	10.5	2.29/1	0	1.0	19.5	34.5	1/1.68
H7321	10	13	14.5	18	2.08/1	0	0.5	14.5	51.0	1/3.40
H4808	15	14	15.5	11.5	3.87/1	0	1.5	24.5	46.5	1/1.79
M0305	12	14	12.5	14	2.75/1	0	3.0	23.5	42.0	1/1.58
A8639	8.5	16.5	18.5	22	1.98/1	0	0.5	24.0	43.5	1/1.78
H1864	16.5	14	3.5	13.5	2.52/1	—	—	—	—	—
H1296	10	9.5	7.5	20	1.35/1	0	3.5	25.0	26.5	1/.93
J4226	6	7.5	12.5	15	1.73/1	0	0	12.5	33.5	1/2.68
J8455	8.5	17.0	20.0	14.0	3.84/1	—	—	—	—	—
L6206	7.5	12	23.5	7.5	5.74/1	—	—	—	—	—
M9987	4.5	7	8.5	13	1.54/1	0	1.0	17.5	30.5	1/1.65
P4185	2	7	13	15.5	1.42/1	0	0.5	14.0	46.0	1/3.17
S7808	3	28.5	17	6.5	7.5/1	—	—	—	—	—
S4171	6	19.5	17.5	5.5	7.8/1	0	1.0	18.5	45.5	1/2.33
S4917	3.0	11.0	22.0	16.0	2.25/1	0	0.5	12.5	36.0	1/2.77
T9985	5.5	11.5	21	7.5	5.04/1	—	—	—	—	—
W0218	5	11	15.5	14.5	2.17/1	—	—	—	—	—
W1083	8	12	16.5	10	3.65/1	0	0.5	24.5	41.5	1/1.66

Part B. Defending Company

Subject	B measure				
	Myelocyte	Juvenile	Band	Segmented	Schilling Index
F8576	5.5	7	14.5	13.5	2/1
B5392	2.5	11.5	26.0	17.5	2.29/1
H4368	3.0	9	13.5	28.5	0.89/1
J4476	2.0	7.5	10.5	13.5	1.48/1
L0624	2.0	7.5	25.5	29.5	1.19/1
M2880	3.0	7.5	30.0	16.0	2.53/1
O8834	6.5	12.5	23.0	24.0	1.75/1
P4431	8.0	6.0	21.0	34.0	1.03/1
P0411	1.5	3.0	26.0	23.0	1.33/1
R0371	3.5	5.5	33.0	14.5	2.90/1
S4749	1.0	4.5	25.5	17.5	1.78/1
T1483	3.0	6.0	23.0	14.0	2.29/1
W7805	3.0	9.5	20.0	20.0	1.63/1

Part C. Control Group (Ft Knox)

Subjects[a]	Segmented[b]	Band	Schilling Index
Wright	47	15	1/3.1
Bright	38	12.8	1/3.0
Price	59.5	5	1/11.9
Musser	43	9.7	1/4.4
Madigan	32.5	13.2	1/2.4
Lehmann	52.5	8	1/6.6
Harp	46.5	12	1/3.9
Anderson	60.5	7	1/8.7
House	40.5	2.7	1/14.5
Loizos	34.7	10	1/3.5
Barnett	39	5.7	1/6.7
Smith	46.8	11.5	1/4.1
Holbrook	38	12.5	1/3.0
Smouse	45.3	7.7	1/5.9
Alexsy	52.7	6.3	1/8.4
Carswell	42.5	10.2	1/4.1
Walmer	46.7	12	1/3.9
Gandey	45.7	3.8	1/12.4

[a]Subjects tested at Ft Knox, 11 March 1953.
[b]Myelocyte and Juvenile cells were absent.

Appendix B

PSYCHOLOGICAL MEASUREMENT

CONTENTS

INTRODUCTION

If drastic physiological changes are observed then psychological changes are expected; if there are changes in neural function, metabolic activity, and adrenal hormone secretion, then such phenomena as reduced capacity to perform, feelings of tiredness, etc. are expected. This area, the behavioral or psychological changes, is particularly important to the study of stress from a military view. It is important that measures be available to determine what aspects of the soldier's behavior are affected by combat and to what extent they are affected. Such a determination presumes that these behavioral changes are evaluated with respect to combat effectiveness and to the job the rifleman must perform.

Whereas physiological indices reduce to a minimum problems of motivation, interest, learning, and intelligence, and are readily quantifiable, some of the techniques require elaborate analysis of the samples.

Refinement of psychological techniques for simpler administration and analysis, in order to reflect the behavioral status of the soldier, is highly desirable. Currently, psychological indices of stress are few and conflicting; consequently it was difficult to select a battery of existing tests to take into combat which would evaluate the effects of battle on the behavior of soldiers.

Tests suggested at the ORO symposium in 1952 were considered,[1] the literature was searched,[4] and informal conferences were held. In some instances, laboratory and ZI field work lent credence to the selection of measures (e.g., visual flicker fusion); at the other extreme recent research suggested new measures, which had never been applied to field studies but which seemed particularly appropriate for this type of study (e.g., auditory flutter fusion). The necessary gamble was recognized but it was hoped that future research would help interpretation of the results. Some tests were eliminated because of elaborateness of equipment; some because of learning and practice effects involved (motor skill tasks). Others involved unrealistic time for administration and inadequate quantification (projective techniques); while some tests were dropped with the foreknowledge that adequate control conditions would not be available (psychogalvanic response, muscle potentials). Other measures, which do not give a generalized picture of the individual's condition, or were liable to controversial results [e.g., electroencephalograph (EEG), tremor, dynamometer] were considered but not included. Finally, detailed analysis of comments by buddies and officers, or the individual's report of how he acts and feels before, during, and after combat would have required doubling the size of the research team. The battery of measures finally chosen represented an attempt to assess the state of cortical sensitivity, and "higher mental functioning." The tests are listed on the following pages.

If the central nervous system is the first to be adversely affected by stress, the higher mental functions should show these detrimental effects.[1] Related work in the area of brain-damaged patients, and neurosurgical therapy lends support to this hypothesis.[38] The most serious common alteration resulting from damage to the frontal lobe is defective judgment.[1(84)] In general the stress of combat probably impairs performance in such areas as timing, accuracy, and patterning of response and also results in the breakdown of integration of functions, memory for recent events, and loss of insight.[3(35)]

MEASUREMENT RESULTS

Higher Mental Function

A preliminary analysis of the results on the psychological tests revealed no effect of stress on performance of the tests of "higher mental functions." If anything, the mean of the scores made by subjects in the Attacking and Defending Companies immediately following combat (B) was slightly higher than the means of the Control Group on first administration (A). In the Attacking and Defending Companies the poststress (B) scores were lower than the scores obtained on second administration several days later (C). There was a similar increase, however, on second administration (A') in the Control Group, undoubtedly due to practice. Thus a higher score upon recovery than immediately following stress could very likely be due to the effects of practice.

Practice effects were anticipated and duplicate forms of the tests were used whenever possible. Even so there appeared to be a consistent gain on second administration, although Form I of parts of the Wechsler-Bellevue Intelligence Scale was always administered first.[39] The Control Group was given the tests as an additional precaution.

Of four of the most promising tests, additional statistical analysis was performed in an attempt to define some of the problems involved in the generally negative psychological results.* The four tests were Digit Symbol, Similarities and Digit Span (all parts of the Wechsler-Bellevue Intelligence Scale), and the Identical Forms test. A description of these tests may be found in ORO-T-41 (FEC).

Since several parts of the Wechsler-Bellevue and other intelligence measures were used, the question may be asked—do the levels of intelligence result in a differential response to stress? The only available estimate of the subject's intelligence was Aptitude Area I of the Army Classification Battery. This score is a composite of scores on the Reading and Vocabulary, Arithmetic Reasoning, and Pattern Analysis Tests.

As shown in Table B1, the Attacking Company has a significantly higher mean Aptitude Area I (AAI) score than the Control Group. There is no significant difference between Attacking and Defending Companies or between the Defending Company and the Control Group.

* The complete battery of tests used in Korea to assess the effect of combat stress on "higher mental functions" comprised: Identical Forms, Cattel Culture Free, Shipley-Hartford Institute of Living Scale, Gottschaldt Figures, Stroop Ratio, Time Estimation, Card Sort, and parts of the Wechsler-Bellevue Intelligence Scale (Digit Symbol, Digit Span, and Similarities). Because of the requirements of the field situation not all the tests were administered according to the standards set by the authors. Details of the test administration are given in ORO-T-41(FEC).

As shown in Table B2 there is a consistent positive correlation, though not always significant, of psychological test scores with Aptitude Area I. In the Control Group the Digit Symbol, Identical Forms, and Digit Span tests show significant correlations with Aptitude Area I scores whereas the Similarities test does not. For the control measures (C) on the stress groups there is some apparent relation, though not as marked. With such small samples and with some nonsignificant correlations it is difficult to establish the relation. The relation shown between the psychological test scores and the Aptitude Area I intelligence estimate of the subjects might be expected.

TABLE B1

ARMY CLASSIFICATION BATTERY, APTITUDE AREA I

Means compared	Mean diff.	t	df.	P
Attacking Co (92.1) vs Defending Co (85.1)	7.0	1.08	45	NS
Attacking Co (92.1) vs Control Grp (81.3)	10.8	2.12	56	<.05
Defending Co (85.1) vs Control Grp (81.3)	3.8	0.54	31	NS

TABLE B2

CORRELATION OF APTITUDE AREA I SCORES WITH PSYCHOLOGICAL TESTS ON CONTROL DATA

Groups	Digit Symbol			Similarities			Identical Forms			Digit Span		
	N	Rho with AAI	P[a]	N	Rho with AAI	P[a]	N	Rho with AAI	P[a]	N	Rho with AAI	P[a]
Control Grp A	22	.69	.01	22	.37	NS	22	.55	.01	22	.54	.01
Attacking Co C	11	.40	NS	10	.92	.01	11	.37	NS	11	.53	.05
Defending Co C	11	.64	.05	11	.29	NS	11	.81	.01	11	.09	NS

[a]Significance based on conversion of rho to r (J. P. Guilford, *Psychometric Methods*, Table 63, p 341, McGraw-Hill Book Co, Inc., New York, 1936) and entrance of df. and r (*ibid.*, Table K, p 548.)

The problem to be examined, however, is whether there is a differential effect of stress (as measured by the test battery) as a function of intelligence (as measured by Aptitude Area I scores). If there is a differential effect it is expected that there would be a greater or lesser suppression of the test scores at the stress measurement, depending on the intelligence level. Thus there would be an expected greater difference between the stress test and the recovery test score.

Table B3 presents the results of correlating the difference between scores on first and second administration of the four tests selected with the Aptitude Area I scores. The tests were administered to the Control Group twice, 11 days apart; these soldiers were in regimental reserve. The stress groups (the Attacking and Defending Companies) first took the tests some 7 to 17 hours after

leaving active combat. The second administration came 5 and 11 days after combat for Attacking and Defending Companies, respectively. It can be seen that there is no apparent significant relation between Aptitude Area I scores and the difference in test scores for the Attacking and Defending Companies, indicating that stress did not affect intelligence levels differently.

TABLE B3

CORRELATION OF APTITUDE AREA I SCORES WITH DIFFERENCE SCORES OF PSYCHOLOGICAL TESTS
(Second Administration Minus First Administration)

Groups	Digit Symbol		Similarities		Identical Forms		Digit Span	
	N	Rho with AAI	N	Rho with AAI	N	Rho with AAI	N	Rho with AAI
Control Grp	15	-.36	15	.13	15	-.06	15	.26
Attacking Co	11	-.33	9	-.04	11	-.40	10	.15
Defending Co	11	.45	11	-.30	11	.47	11	.36

TABLE B4

PSYCHOLOGICAL TEST SCORES—MEAN OF DISTRIBUTION OF DIFFERENCES[a]

Groups	Identical Forms		Digit Symbol		Digit Span		Similarities	
	N	Mean	N	Mean	N	Mean	N	Mean
Control Grp A'-A	16	3.8	16	1.19	18	-.11	18	.78
Attacking Co C-B	11	3.0	11	10.10	10	-.80	9	.44
Defending Co C-B	12	3.2	12	5.00	12	1.08	12	-.75

[a]Each individual's score on the first administration of the test was subtracted from his score on the second administration, and the mean of these differences was computed for the group.

Welford et al.[40] describe an effect where the score of first and second administration of a test is a function of the subject's condition when the test is first taken. Following this it would be expected that the Control Group might show a gain on the second administration since the subjects were "fresh" when they first encountered their task, and any normal practice on learning effects should have appeared on the second administration. On the other hand the stress groups first encountered the tests following combat, and according to Welford et al. this would affect the second administration so that little or no gain would be shown.

From Table B4 it is clear that no such pattern resulted. The Control Group does show a higher mean gain on the Identical Forms and Similiarities tests, but it is very small and not significant. The two other tests, Digit Symbol and Digit Span, in general present the opposite picture, in that the mean gain is higher for the stress groups. The Attacking Company mean

gain on Digit Symbol is significantly higher than the mean gain of the Control Group (t = 3.58, P = .01) or the Defending Company (t = 2.71, P = .02). The mean gain of the Defending Company on Digit Span is significantly higher than that of the Attacking Company (t = 2.52, P = .02).

It might seem from Table B4 that the scores on the first administration of the tests to the stress groups were depressed for some reason. Yet if this were true the means in Table B5 for the stress groups' first administration should be lower than that of the control groups. The converse is true, for the means are higher in Defending and Attacking Companies, in two instances significantly [Attacking Company means are significantly higher on Identical Forms (t = 2.13, P = .05) and Digit Span (t = 2.23, P = .05)].

TABLE B5

MEANS OF PSYCHOLOGICAL TEST SCORES ON FIRST ADMINISTRATION

Groups	Identical Forms		Digit Symbol		Digit Span		Similarities	
	N	Mean	N	Mean	N	Mean	N	Mean
Control Grp	24	11.4	24	34.8	24	9.79	24	10.3
Attacking Co	12	14.1	12	36.7	10	11.40	10	10.0
Defending Co	13	12.2	13	37.1	13	10.10	13	11.8

These results are confounded by the fact that Attacking and Defending Companies had a higher intelligence level than the Control Group (see Table B1) and there is some correlation of the tests with intelligence (see Table B2). In addition the administrators felt that Attacking and Defending Companies were better motivated to take the tests than the Control Group.

In spite of the fact that the psychological tests showed no effect of combat stress, there still might have been some relation between these tests and the physiological measures. Using the Digit Symbol test, correlations were run between scores on this test and two of the physiological measures, as well as change in these measures from stress to recovery. Correlations were also run between estimates of combat performance (in the form of ratings by the team psychiatrist and company officers) and the Digit Symbol test scores.

These results are presented in Table B6. There is a striking lack of correlation between the test scores and any of the other measures.

The results of this analysis support the conclusions drawn in the earlier report, ORO-T-41 (FEC), i.e., that these tests of higher mental functions failed to demonstrate a significant change with stress and did not appear to be related to the other measures. Subsequent work was done by the Adjutant General's Office on psychological tests used in the stress situation of the paratroop trainees' 34-foot jump tower. The Digit Symbol, Digit Span, and other mental ability tests used generally showed no stress effects.[41]

Sensory-Cortical Sensitivity

A description of the use of visual flicker fusion (VFF) and auditory flutter fusion (AFF) as measures of sensory-cortical sensitivity were given in ORO-

TABLE B6

CORRELATION[a] OF DIGIT SYMBOL TEST
WITH PHYSIOLOGICAL MEASURES AND RATINGS

Physiological measure	Group	Digit Symbol Test: Attacking Co, B	Attacking Co, C score minus B score	Defending Co, B	Defending Co, C score minus B score	Control Group, A	Control Group, A' score minus A score
Urine Na/K ratios	Attacking Co, B	N = 12 r = −.03					
	Attacking Co, C minus B		N = 11 r = .16				
	Defending Co, B			N = 13 r = −.25			
	Defending Co, C minus B				N = 12 r = −.05		
	Control Grp, A					N = 24 r = .13	
	Control Grp, A' minus A						N = 16 r = .16
17 KS, mq/hr	Attacking Co, B	N = 12 r = −.10					
	Defending Co, B			N = 12 r = .01			
	Attacking Co, C minus B		N = 11 r = −.39				
	Defending Co, C minus B				N = 11 r = .38		
	Control Grp, A					N = 24 r = −.01	
Company ratings	Psychiatrist's ratings Attacking Co	N = 12 r = .66					
	Sergeant's ratings, Attacking Co	N = 12 r = −.01					
	Psychiatrist's ratings, Defending Co			N = 12 r = −.02			
	Officer's ratings, Defending Co			N = 12 r = −.11			

[a]Significance based on conversion of rho to r (J. P. Guilford, *Psychometric Methods*, Table 63, p 341, McGraw-Hill Book Co, Inc., New York, 1936) and entrance of df. and r (*ibid.*, Table K, p 548). None of the values are significant.

panies were combined and treated as one group. Subsequently the two groups have been found to respond differently on many of the measures; therefore, in Tables B7 and B8 results on VFF and AFF are presented separately for these companies.

TABLE B7

COMPARISON OF GROUP MEANS ON VFF AND AFF, ATTACKING COMPANY

Means compared	Mean diff.	t	df.	P
VFF, cpm				
Attacking Co B (2608) vs Attacking Co C (2839)	231	4.88	10	<.001
Attacking Co B (2582) vs Attacking Co D (2825)	243	4.26	8	<.01
Attacking Co C (2820) vs Attacking Co D (2825)	5	—	—	NS
Attacking Co B (2608) vs Control Grp A[a] (2698)	90	1.38	33	NS
Attacking Co C (2839) vs Control Grp A[a] (2698)	141	2.31	33	<.05
AFF, cps				
Control Grp A (30.7) vs A′ (36.7)	6	2.54	17	<.05
Attacking Co B (27) vs Attacking Co C (33.36)	6.36	3.02	10	<.02
Attacking Co B (26.4) vs Attacking Co D (32)	5.6	1.95	9	NS
Attacking Co C (33.3) vs Attacking Co D (32)	1.3	—	9	NS

[a]No A′ data were available on the Control Group owing to equipment failure.

TABLE B8

COMPARISON OF MEANS ON VFF AND AFF, DEFENDING COMPANY

Means compared	Mean diff.	t	df.	P
VFF, cpm				
Defending Co B (2658) vs Defending Co C (2732)	74	1.07	11	NS
Defending Co B (2636) vs Control Grp A (2698)	62	0.90	35	NS
Defending Co C (2732) vs Control Grp A (2698)	34	0.53	34	NS
AFF, cps				
Defending Co B (22.7) vs Defending Co C (29.4)	6.7	2.92	11	<.02
Defending Co B (22.7) vs Control Grp A (30.7)	8.0	1.04	28	NS
C-B Attacking Co (6.36) vs C-B Defending Co (6.36)	0.56	—	21	NS

In general, the stressed groups show a lower mean fusion frequency on both AFF and VFF a few hours following combat than on their measures several days following combat. The Control Group likewise shows a significantly

*Visual flicker fusion refers to the point at which an interrupted white light is perceived as being continuous. Auditory flutter fusion refers to the point at which an interrupted white noise is perceived as being continuous. These fusion points have been found to vary according to the condition of the subject in several experimental situations.

men' ailure, only one measure of VFF was available on the Control Group. This measure was not significantly different from the stressed groups' first administration.

It would appear that the results on AFF and VFF could also be accounted for by practice or related effects. As reported in ORO-T-41 (FEC), the preliminary analysis of the data was encouraging. A more detailed analysis demonstrated, however, that although the changes observed might partly be a result of stress they were seriously confounded by variables inherent in the measuring devices. These factors make interpretation of the data impossible at the present time.

The practice effect has been observed by others in VFF measurement.[42,43] There is no similar body of literature pertaining to this problem in AFF. Some additional work by ORO, using AFF as a stress measure, demonstrates rather conclusively the existence of a practice effect which to some extent might mask any stress effects.

In one such study the AFF was administered to a random sample of an armored company engaged in maneuvers at Ft Knox, Ky.[44] The test was administered on four occasions, approximately 10 days apart (before and after two 10-day maneuvers). Differing from the Korean administration of the AFF, where individual means were based on an average of three trials, the AFF at Ft Knox was based on an average of five trials following two practice trials.

The same subjects were involved in the four administrations of the AFF at Ft Knox. The first three administrations were confounded by what might have been a practice effect plus the use of different administrators. The machine is not automatic and consequently manipulation of the controls may vary with administrators. Referring to Table B9, the mean of 33 CPS premaneuver 1 increased to 42 during postmaneuver 1, and increased still further to 45 during premaneuver 2. The mean, postmaneuver 2, dropped to 39, which may indicate a real effect on fusion frequency as a result of a 10-day maneuver.

In addition, ORO participated in the stress experiments conducted at Ft Benning by the Personnel Research Branch of the Adjutant General's Office in July 1953, using 48 paratroopers making their first jump from the 34-foot tower.[41] Subjects were given four practice and four test trials on AFF in both stress and nonstress conditions. In these tests a trial consisted of the subject's adjusting the Repetition Rate dial until he found the point at which he thought the sound was just beginning to be interrupted. In other experiments this had always been done by the experimenter. There was no significant difference between stress and nonstress conditions in the flutter fusion point, but there was a consistent "learning" effect; i.e., the subjects consistently show a higher fusion point in the second series of trials regardless of whether the first administration was under stress or nonstress conditions.[45]

In a further attempt to study the learning or practice effect encountered in these experiments, two groups of 10 subjects each were run. The "continuous" group received 2 practice trials and then 20 consecutive test trials all at one session. The "spaced" group received 2 practice and 10 test trials at the first session, and 10 additional test trials at a second session 24 hours later, for a total of 20 trials. Although the t test of differences was not significant, the data showed that the "spaced" group definitely had a larger increase in cps from the first 10 to the second 10 trials than did the "continuous"

group. These results suggest that the "practice" effect found in the past may be due not only to practice but also possibly to some other factor such as the subjects changing their "criterion" of fusion from one time to the next, or possibly, spacing may be a more effective condition of practice.

TABLE B9

COMPARISON OF AFF[a] MEANS OF MEN PARTICIPATING IN TANK MANEUVERS
(Ft Knox, 1953)
(cps)

Means compared	Mean diff.	t	df.	P
Pre-1[b] (33.1)[c] vs Post-1 (42.4)	9.3	4.48	29	<.001
Pre-1 (32.2) vs Pre-2 (43.8)	11.6	5.67	26	<.001
Pre-1 (33.4) vs Post-2 (38.3)	4.9	3.90	23	<.001
Post-1 (42.1) vs Pre-2 (44.7)	2.6	1.29	27	NS
Post-1 (42.4) vs Post-2 (38.8)	3.6	1.70	24	NS
Pre-2 (43.1) vs Post-2 (38.7)	5.4	3.88	23	<.001

[a]Settings of AFF: on-off ratio 90 percent, attenuation 72 db. Values of t computed from distribution of individual mean differences.

[b]Dates of maneuvers: Pre-1, 11 March; Post-1, 20 March; Pre-2, 31 March; Post-2, 9 April.

[c]Premaneuver 1 was administered by two individuals, 15 subjects each. The inter-administrator means were significantly different: P = .05. Postmaneuver 1, pre- and postmaneuver 2 were administered by a third individual.

Summary

The fact that the tests did not satisfactorily reflect changes caused by combat stress is thought to be primarily a result of measurement problems. The team felt that there were definite changes in the men's behavior (such as an observable depression of activity) as a result of being in combat, but that these changes were not measured because of one or more of the following factors: (a) the tests were not sensitive to the stress; (b) the time lag between stress and testing was too great, permitting recovery; (c) the subjects were motivated to exert compensatory effort at the time of testing; (d) the test conditions were not adequately standardized; and (e) practice effect masked any real deterioration of performance. These factors, coupled with the relatively large amount of time taken to administer the tests to each subject, indicate a great need for developing simple, behavioral indices suitable for field testing. It has been pointed out that where there has been a wealth of basic research on the adrenal functions, there is a gross lack of basic research underlying the area of stress-sensitive psychological tests. Perhaps one of the reasons that the present results have been so discouraging is that the analysis of behavioral processes under stress has not come first.[3(34)]

REFERENCES

1. Operations Research Office, "Fatigue and Stress Symposium," ORO-T-185, 10 Sep 52.
2. Operations Research Office, "A Study of Combat Stress, Korea, 1952 (Preliminary Report)," ORO-T-41 (FEC).
3. Operations Research Office, "Symposium on the Role of Stress in Military Operations," ORO-T-256, 2 Dec 53.
4. Operations Research Office, unpublished notes on the analysis of recent fatigue and stress literature.
5. H. Selye, Stress, Acta, Inc., Montreal, 1950.
6. W. S. Cannon, Bodily Changes In Pain, Hunger, Fear, and Rage, 2d ed.; D. Appleton Century Company, Inc., New York, 1929.
7. G. Pincus, and F. Elmadjian, "Pituitary-adrenal System," Ann. Rev. Physiol., 16:403 (1954).
8. D. J. Ingle, "The Role of The Adrenal Cortex in Homeostasis," J. Endocrin., 8:23 (1952).
9. J. N. Swanson, W. Bauer, and M. Ropes, "Evaluation of Eosinophil Counts," Lancet, 1:129 (1952).
10. Symposium on Stress, Army Medical Service Graduate School, Washington, D. C., 16 - 18 March 1953, pp. 55-57.
11. R. J. Humphreys, and W. Raab, "Response of Circulating Eosinophils to Nonepinephrine, Epinephrine and Emotional Stress in Humans," Proc. Soc. Exp. Biol., N. Y., 74:302 (1950).
12. K. Tatai, Y. Mori, and K. Ito, "Response of the Pituitary-adrenocortical System to Mental Strain in Healthy Women," Jap. J. Physiol., 1:316 (1951).
13. T. J. Domanski, A. G. Swan, J. G. Wells, and L. B. Hughes, "Physiological Relationships in Human Stress Response. I: Eosinophil Response to Muscular Activity," USAF, Sch. Aviat. Med., Project No. 21-32-025, Rept. No. 1, Oct 51, pp. 1-13.
14. J. W. Frost, R. L. Dryer, and K. G. Kohlstaedt, "Stress Studies on Auto Race Drivers," J. Lab. Clin. Med., 38:523 (1951).
15. A. E. Renold, T. B. Quigley, H. E. Kennard, and G. W. Thorn, "Reaction of the Adrenal Cortex to Physical and Emotional Stress in College Oarsmen," New Eng. J. Med., 244:754 (1951).
16. J. M. Adam, and W. S. S. Ladell, "Report of Field Studies on Troops of the Commonwealth Division in Korea, Winter 1951-52," Great Britain, Dept. of Scientific Adviser to the Army Council. (British-RESTRICTED, US-CONFIDENTIAL)
17. H. Hoagland, "Metabolic and Physiologic Disturbances in the Psychoses," The Biology of Mental Health and Disease, pp. 434-449, Harper & Brothers, New York, 1952.

18. H. Hoagland, F. Elmadjian, and G. Pincus, "Stressful Psychomotor Performance and the Adrenal Cortical Function as Indicated by the Lymphocyte Response," J. Clin. Endocrinol., 6:301 (1946).
19. W. J. Kuhl, H. Wilson, and E. P. Ralli, "Measurements of Adrenal Cortical Activity in Young Men Subjected to Acute Stress," J. Clin. Endocrinol. Metab., 12:393 (1952).
20. J. W. Conn, W. C. Vogel, L. H. Louis, and S. S. Fajans, "Serum Cholesterol: A Probable Precursor of Adrenal Cortical Hormones," J. Lab. Clin. Med., 35:504 (1950).
21. J. M. Tanner, "The Relation Between Serum Cholesterol and Physique in Healthy Young Men," J. Physiol., Lond., 115:371 (1951).
22. H. Hoagland, Chapter XXI: "Stress and the Adrenal Cortex with Special Reference to Potassium Metabolism," in Life Stress and Bodily Disease, vol. XXIX, 1950.
23. G. W. Thorn, D. Jenkins, and J. D. Laidlaw, "The Adrenal Response to Stress in Man, in Recent Progress in Hormone Research, vol. 8, pp. 171-215, Academic Press, Inc., New York, 1953.
24. G. Pincus, H. Hoagland, H. Freeman, F. Elmadjian, and L. P. Romanoff, "A Study of Pituitary-adrenocortical Function in Normal and Psychotic Men," Psychosom. Med., 11:74 (1949).
25. A. Forbes, The 17 Ketosteroid Excretion in Stress. Preliminary Report. Josiah Macy Jr. Foundation Report, New York, 1942.
26. H. L. Mason and W. W. Engstrom, "The 17 Ketosteroids; Their Origin Determination and Significance," Physiol. Rev., 30:321 (1950).
27. A. P. Forbes, E. C. Donaldson, E. C. Reifenstein, and F. Albright, "The Effect of Trauma and Disease in the Urinary 17 Ketosteroid Excretion in Men," J. Clin. Endocrinol., 7:264 (1947).
28. H. Hoagland, "Adventures in Biological Engineering," Science, 100(2587) :63 (1944).
29. G. Pincus and H. Hoagland, "Steroid Excretion and the Stress of Flying," J. Aviat. Med., 14:173 (1943).
30. E. B. Cook and R. J. Wherry, "The Urinary 17 Ketosteroid Output of Naval Submarine Enlisted Candidates During Two Stressful Situations," Hum. Biol., 22:104 (1950).
31. G. Pincus, "The Analysis of Human Urines for Steroid Substances," J. Clin. Endocrinol., 5:291 (1945).
32. W. J. Reddy, D. Jenkins, and G. W. Thorn, "Estimation of 17 Hydroxycorticoids in Urine," Metabolism, 1:511 (1952).
33. G. Pincus, "A Diurnal Rhythm in the Excretion of Urinary Ketosteroids by Young Men," J. Clin. Endocrinol., 3:195 (1943).
34. H. M. Grundy, S. A. Simpson, and J. F. Tait, "Isolation of a Highly Active Mineralocorticoid from Beef Adrenal Extract," Nature, 169:795 (1952).
35. Browne, Hoffman, Schenker, Venning, and Weil, "Metabolic Aspects of Damage and Convalescence in Acutely Injured Persons, Contrasting Previously Healthy Subjects with Previously Debilitated Patients," in Conference on Metabolic Aspects of Convalescence Including Bone and Wound Healing, E. C. Reifenstein, Jr., (ed.) Ninth Meeting, Feb 2-3, sponsored by the Josiah Macy, Jr., Foundations, New York, 1945.

36. G. Pincus, E. Romanoff, and J. Cahn, "Excretion of Urinary Steroids by Men and Women of Various Ages," J. Geront., 9:113 (1954).
37. E. M. Glenn, and L. H. Nelson, "Chemical Method for the Determination of 17 Hydroxycorticoids and 17 Ketosteroids in Urine Following Hydrolysis with Beta-glucuronidase," J. Clin. Endocrinol. Metab., 13:911 (1953).
38. F. A. Mettler (ed.), Selective Partial Ablation of the Frontal Cortex, Paul B. Hoeber, Inc., Medical Book Dept. of Harper & Brothers, New York 1949.
39. R. Gerboth, "A Study of the Two Forms of the Wechsler-Bellevue Intelligence Scale," J. Consult. Psychol., 14:365 (1950).
40. A. T. Welford, R. A. Brown, and J. E. Gabb, "Two Experiments on Fatigue as Affecting Skilled Performance in Civilian Air Crew," Brit. J. Psychol., 40:195 (1950).
41. J. G. Miller, "The Development of Experimental Stress Sensitive Tests for Predicting Performance in Military Tasks," Personnel Research Branch, Adjutant General's Office, Report 1079, October 1953.
42. O. W. McNemar, "The Ordering of Individuals in Critical Flicker Frequency Under Different Measurement Conditions," J. Psychol., 32:3 (1951).
43. C. Landis, "Determinants of the Critical Flicker Fusion Threshold," Physiol. Rev., 34:259 (1954).
44. Operations Research Office, unpublished notes on the organizational test of a tank company equipped with the T48 tank.
45. Operations Research Office, unpublished notes on testing and use of certain stress-sensitive measures.